# CLINICAL AND ANGIOGRAPHIC CHARACTERISTICS OF PATIENTS WITH COMBINED ANTERIOR AND INFERIOR ST-SEGMENT ELEVATION ON THE INITIAL ELECTROCARDIOGRAM DURING ACUTE MYOCARDIAL INFARCTION

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**THE TAMILNADU**
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# CERTIFICATE

This is to certify that the dissertation entitled **– "CLINICAL AND ANGIOGRAPHIC CHARACTERISTICS OF PATIENTS WITH COMBINED ANTERIOR AND INFERIOR ST SEGMENT ELEVATION ON THE INITIAL ELECTROCARDIOGRAM DURING ACUTE MYOCARDIAL INFARCTION"** is the bonafide original work of **Dr. A. SRINIVASAN** in partial fulfillment of the requirements for D.M.Branch II (CARDIOLOGY) examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held on August 2013. The period of post graduate study and training was from August 2010 to July 2013.

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# DECLARATION

I **Dr. A. SRINIVASAN**, solemnly declare that this dissertation entitled **– "CLINICAL AND ANGIOGRAPHIC CHARACTERISTICS OF PATIENTS WITH COMBINED ANTERIOR AND INFERIOR ST SEGMENT ELEVATION ON THE INITIAL ELECTRO CARDIOGRAM DURING ACUTE MYOCARDIAL INFARCTION"** is the bonafide original work done by me at the Department of Cardiology, Stanley Medical College and Government Stanley Hospital during the period 2010-1013 under the guidance and supervision of the Professor and Head of Department of Cardiology of Stanley Medical College and Government Stanley Hospital, **Prof. Dr. K.KANNAN, M.D., D.M.** This dissertation is submitted to THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY, towards partial fulfillment of requirement for the award of D.M. Degree (Branch - II) in cardiology.

Place : **Dr. A .SRINIVASAN**

Date :

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# ABBREVIATIONS

| | | |
|---|---|---|
| 2D | - | Two Dimensional |
| 3D | - | Three Dimensional |
| AMI | - | Acute Myocardial Infarction |
| AWMI | - | Anterior Wall Myocardial Infarction |
| AV | - | Atrioventricular |
| BMI | - | Body Mass Index |
| BP | - | Blood Pressure |
| BSA | - | Body Surface Area |
| CABG | - | Coronary Artery By-Pass Graft |
| CAD | - | Coronary Artery Disease |
| CAG | - | Coronary Angiogram |
| CD | - | Cluster Designation |
| CHF | - | Congestive Heart Failure |
| CI | - | Confidence Interval |
| CPK | - | CreatininePhospho Kinase |
| CT | - | Computed Tomography |
| CVS | - | Cardio Vascular System |
| D1 | - | first diagonal artery |
| df | - | difference of freedom |
| DNA | - | Deoxyribo Nucleic Acid |
| DT | - | Deceleration Time |
| DVD | - | Double Vessel Disease |
| ECG | - | Electrocardiogram |
| HBsAg | - | Hepatitis B surface Antigen |
| HT | - | Height |
| ICCU | - | Intensive Coronary Care Unit |
| IRA | - | Infarct Related Artery |
| IWMI | - | Inferior Wall Myocardial Infarction |

| | | |
|---|---|---|
| LAD | - | Left Anterior Descending artery |
| LCX | - | Left Circumflex artery |
| LMCA | - | Left Main Coronary Artery |
| LV | - | Left Ventricle |
| LVEDD | - | Left Ventricular End Diastolic Dimension |
| LVEF | - | Left Ventricular Ejection Fraction |
| LVESD | - | Left Ventricular End Systolic Dimension |
| MI | - | Myocardial Infarction |
| MR | - | Mitral Regurgitation |
| NADH | - | Reduced form of Nicotinamide Adenine Dinucleotide |
| NADPH | - | Reduced form of Nicotinamide Adenine Dinucleotide Phosphate |
| NF-kB | - | Nuclear factor Kappa-B Ligand |
| PCI | - | Percutaneous Coronary Intervention |
| PDGF | - | Platelet Derived Growth Factor |
| PR | - | Pulse Rate |
| RANKL | - | Receptor Activator of Nuclear factor Kappa-B Ligand |
| RCA | - | Right Coronary Artery |
| RS | - | Respiratory System |
| RV | - | Right Ventricle |
| S1 | - | first septal artery |
| SCAI | - | Subcommittee on percutaneoustransluminal coronary angioplasty |
| SD | - | Standard Deviation |
| SMC | - | Smooth Muscle Cell |
| SVD | - | Single Vessel Disease |
| TDI | - | Tissue Doppler Imaging |
| TGF-β | - | Transforming Growth Factor beta |
| TVD | - | Triple Vessel Disease |
| VSR | - | Ventricular Septal Rupture |
| WMSI | - | Wall Motion Score Index |
| WT | - | Weight |

# INTRODUCTION

Myocardial infarction is the common disease nowadays and ST elevation myocardial infarction is important one among the ACS(Acute Coronary Syndrome). It is quite rare a situation when patient presents with simultaneous ST elevation in both anterior and inferior leads during acute myocardial infarction. In this thesis, we are going to see the various implications of this rare presentation in myocardial involvement as assessed by electrocardiogram and distribution of lesions in coronary arteries as assessed by coronary angiogram. The life- saving and crucial use of reperfusion therapy for acute myocardial infarction (AMI)[1] makes it important to find out the location of the coronary arterial lesion responsible for the infarct prior to initiating treatment.[2]In anterior AMIs, location of the lesion in the anterior descending artery (AD) has been associated with the amount of myocardial necrosis and prognostic events.[3,4] An acute obstruction of the coronary artery causes extensive necrosis that is most oftenco-existed by hemodynamicdeterioration.[2] Therefore, localization of the coronary arterial lesion is useful in evaluating myocardium at risk and in selecting the therapeutic strategy to be used.[5]Recently, many studies in the medical literature find out which features of electrocardiography mainly spot theculprit arteryresponsible for the AMI and the location of the coronary arterial lesion.[2,5-14]

## DIAGNOSIS OF MYOCARDIAL INFARCTION:[15]

### Diagnosis of Myocardial Infarction by Different Techniques

| TECHNIQUE | FEATURES |
|---|---|
| Pathology | Death of Myocardial cell |
| Biochemistry | Markers of myocardial necrosis detected from blood samples |
| Electrocardiography | Evidence of ischemic myocardium (ST and T wave abnormalities); Evidence of absence of electrically functioning cardiac tissue (Q waves) |
| Imaging | Absence or Reduction of myocardial tissue perfusion; Regional wall motion abnormalities of myocardium |

# REVIEW OF LITERATURE

**SUBHEADINGS:**

PATHOGENESIS OF ATHEROSCLEROSIS AND AMI

ISCHEMIA CASCADE

INFARCT SITE SIZE SAVAGE INDEX SCORES

ROLE OF ECG IN AMI

ROLE OF 2D ECHO IN AMI

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CORRELATION OF 2D ECHO AND AMI.

CLINCAL FEATURES OF COMBINED AWMI AND IWMI

ECG FEATURES OF AWMI AND IWMI

ECHO FEATURES OF COMBINED AWMI AND IWMI

CLINICAL OUTCOMES OF COMBINED AWMI AND IWMI

## PATHOGENESIS OF ATHEROSCLEROSIS AND AMI:

With eating an atherogenic diet, high in cholesterol and saturated fat, small lipoprotein particles deposited in the tunica intima. Theselipoprotein particles surround the proteoglycan of the tunica intima and coalescing to form aggregates.[16] Studies indicate that the endothelial layer permeability becomes high to low-density lipoprotein (LDL) at the sites of predilection for lesion formation. In the nascent atheroma,NADH/NADPH oxidases present in vascular cells, lipoxygenases present in infiltrating leukocytes, or the myeloperoxidases take part in oxidative stress.

### Evolution of the atherosclerotic plaque:

1. Accumulation of lipoprotein particles in the tunica intima and undergo modifications in the form of oxidation and glycation.
2. Oxidative stress and substances found in modified lipoproteins, can cause cytokines to increase.
3. The increased cytokines cause increased expression of leukocyte adhesion molecules and increased amounts of chemo attractant molecules producing leukocyte attachment and migration into tunica intima.

4. Monocyte chemoattractant protein 1 (MCP-1), attracts monocytes to tunica intima and increase their expression of scavenger receptors due to macrophage colony-stimulating factor.
5. These Scavenger receptors take up the modified lipoprotein particles and predispose to the production of foam cells.These Macrophage foam cells form more cytokines and substances like hypochlorous acid, superoxide anion ($O_2^-$), and matrix metallo proteinases.
6. Smooth Muscle Cells then migrateto the tunica intima from the tunica media.
7. Smooth Muscle Cells then divide and cause formation of extracellular matrix in atherosclerotic plaque. Thus, the fatty streak can become into a fibro fatty lesion.
8. After these occurrences, fibrosis and calcification of lesion happens, occasionally associated with apoptosis of smooth muscle cell, forming an acellular fibrous capsule around a lipid-rich core.

**Leukocyte Recruitment:**

Normally endothelial cell opposes adhesive functions ofleukocytes. In inflammation, most often the accumulation of leukocytes occur not in the arteries, but in the postcapillary venules. In hyperlipidemia, leukocytes attach to the endothelial cell and move in between the gap junctions, or sometimes penetrate endothelial cells, a process called

'transcytosis' to enter tunica intima, where they accumulate lipids and become foam cells.[17] Two major divisions of leukocyte adhesion molecules include vascular cell adhesion molecule 1 (VCAM-1) or CD106 which interact very late antigen 4(VLA-4) found in the monocytes and T cells of nascent atheroma.[20] Among the adhesion molecules, there are P-selectins which attract leukocytes on endothelium over atheroma and E-selectins which act as attractants for polymorpho nuclear leukocytes. Selectins cause saltatory or rolling movement of leukocytes over the endothelium.

There are two important chemokines in leukocyte recruitment. One is monocyte chemo attractant protein 1 (MCP-1), produced by the endothelium in response to oxidized lipoprotein.[21] The other one is Interleukin-8, a chemokine that binds to CXCR2 on leukocytes. Fractalkine, another chemokine, favours atherogenesis. Interferon-γ, a cytokine presentin atheromatous plaques, stimulates the T-cell chemo attractants encoding genes.[23,24]

**Intracellular Lipid Accumulation: Foam Cell Formation:**

The scavenger receptors mediate the excessive lipid uptake peculiar of foam cell formation and the receptors responsible for foam

cell formation include CD36 and macrosialin which binds oxidised LDL.[26,27]

Granulocyte-Macrophage Colony-Stimulatory Factor (GM-CSF), Macrophage Colony-Stimulating Factor (M-CSF) and Interleukin-3 present in human atheromatous lesions favour division of macrophages and foam cell formation.

**Smooth Muscle Cell Migration and Proliferation:**

SMCs of the normal arterial tunica media differ significantly from the SMCs found in the intima of an evolving atheroma.[31,32] Platelet-derived growth factor (PDGF) is a powerful SMC chemo attractant which is secreted by activated macrophages and found in increased amounts in human atherosclerosis. There is an 1% multiplication rate for smooth muscle cells in tunica intima and even such low rate of multiplication can lead to significant SMC accumulation in the atheromatous lesions.

Rupture of atheromatous plaques lead to exposure of smooth muscle cells to thrombin and some mitogens which further lead to active migration and replication of SMCs in lesions.[28,29]

**Apoptosis of Smooth Muscle Cell in Atherogenesis**:

Inflammatory cytokines in atheroma and soluble cytokines trigger apoptosis or programmed cell death in smooth muscle cells(SMCs). Fas ligand on the surface of T cells found in atheromatous lesions bind Fas on the surface of SMCs and along with soluble proinflammatory cytokines lead to apoptosis of SMCs as demonstrated microscopically by fragmented nuclear DNAs.[30]

**Angiogenesis in Plaques:**

Angiogenetic factors present in atheromatous plaques include vascular endothelial growth factor (VEGF), placental growth factor (PlGF), and oncostatin M and they lead to angiogenesis in plaques and the occurrence of which provide large surface area for leukocyte migration, growth of plaques and increased perfusion of lesions which in turn lead to further growth of lesions by supplying angiogenetic factors.

**Plaque Mineralization:**

Virchow identified morphologic features of bone formation in microscopic examination of atheromatous plaques. Receptor activator of NF-κB ligand (RANKL) induces mineral formation through a pathway dependent on bone morphogenetic protein- 4 (BMP-4) in SMCs.

Osteoprotegerin can antagonize plaque mineralization by inhibiting RANKL signaling. Oxidative stress and inflammation factors activate a transcription factor called Runx-2, which promotes SMC mineral formation by activatingProtein Kinase B.[34,35]

**Plaque Rupture and Thrombosis:**

Most of myocardial infarctions do not result from high-grade stenoses but from lesions that, often less than 50% stenosis. Only 15% of myocardial infarctions result from lesions of more than 60% stenosis on previous angiogram.

Two thirds of acute myocardial infarctions result from plaque rupture which is often due to plaque's thin fibrous cap fracture. About one quarter of acutemyocardial infarctions result from superficial erosion of the intimal layer of atheromatous lesion and this occurs most often in women than in men as a cause of coronary sudden death and in individuals with hypertriglyceridemia and diabetes mellitus.[36]

The rupture of the plaque's thin fibrous cap is due to an imbalance between the factors that affect the plaque's cap and the factors that increase the integrity of the fibrous cap like the T cell–derived cytokine interferon-γ which inhibits SMC collagen synthesis versus the substances that are released from platelet granules during activation (including TGF-

β and PDGF) which increase SMC collagen synthesis and increase the strength of the plaque's fibrous cap.[37]

**Thrombosis due to Superficial Erosion of Plaques:**

Apoptosis of endothelial cells frequently lead to desquamation of endothelial cells in areas of superficial erosion. Certain gelatinases degrade the basement membrane collagen (e.g., collagen type IV), disintegrate the endothelial cell from the underlying basal lamina and pave path to their desquamation. Repetitive cycles of plaque rupture with subsequent in situ thrombosis and healing causes evolution of the lesionand plaque growth and these events eventually lead to SMC proliferation, migration and matrix synthesis. Studies indicate that the non occlusive thrombosis usually lead to death.[38,39]

**ISCHAEMIA CASCADE:**

When complete occlusion of a coronary artery occurs, hypokinesis of myocardium occurs within 20 seconds and ST elevation occurs after 20 seconds. Angina occurs after 40 seconds of complete occlusion of coronary artery. Diastolic dysfunction occurs earlier than systolic dysfunction in ischemia. The overall pattern of these ischemic events is called ischemic cascade.[40]

## ROLE OF ECG IN MI:

With myocardial infarction, depolarization (QRS) changes and repolarization (ST-T) changes occur in ECG. Myocardial necrosis produces decreased R wave amplitude or Q waves in the respective leads of the affected myocardial area due to loss of electromotive forces. Studies have shown that transmural infarcts can present without Q waves and sub endocardial infarcts can present with Q waves.[41,42] Accordingly, infarcts can be conveniently classified as Q wave or non–Q-wave rather than as transmural or non transmural, based on the ECG.

### Evolution of Electrocardiographic Changes:

ST-segment elevation and hyperacute T wave changes occur as the early changes of ST Elevation MI followed by evolving T wave inversion and sometimes Q waves in the same lead distribution. The T wave inversions either resolve after days or weeks, or persist indefinitely. The infarct size may be an important determinant of T wave evolution. One study showed that cases with transmural infarction and myocardial fibrosis showed that T waves were negative for more than 1 year in leads having Q wave and cases of non transmural infarction have positive T waves in leads with Q waves that correlated with viable myocardium within the wall.[43]

When the left ventricular ejection fraction and regional wall motion improve in cases of smaller area myocardial infarction, the ECG may become a normal one which is associated with spontaneous recanalization or an effective collateral circulation. But the Q waves and ST-segment elevation which persist for weeks to months after an MI indicate an underlying severe wall motion abnormality either akinesis or dyskinesis with or without ventricular aneurysm. The presence of an rSR′ in the mid and left sided chest leads or in lead I is also an ECG marker of ventricular aneurysm.[44]

Some patients with anginal chest pain have deep T wave inversions in V1-V4, with or without elevation of cardiac biomarkers. This is caused by high-grade stenosis in the proximal left anterior descending (LAD) coronary artery (also called as the LAD-T wave pattern or Wellens T waves).

Transient ST-segment elevation that resolves by the time when the patient comes to the physician is the usual antecedent event in this case. These T wave inversions suggest myocardial stunning syndrome if associated with unstable angina and hypokinesis of anterior segments of left ventricle and this syndrome has high incidence of recurrent angina and myocardial infarction. If patients with baseline T wave inversion

develop acute transmural ischemia, paradoxical T wave normalization (pseudonormalization) can occur.

**Localization of Ischemia or Infarction:**

ST-segment elevation and/or hyperacute T waves are present in the following:

1. Two or more contiguous leads ($V_1$ to $V_6$) and/or leads I and aVL with acute anterior or anterolateral wall infarction
2. Leads $V_1$ to $V_3$ with anteroseptal or apical[45] infarction
3. Leads $V_4$ to $V_6$ with apical or lateral infarction
4. Leads II, III, and aVF with inferior wall infarction
5. Right-sided precordial leads V3R, V4R, V5R with right ventricular infarction.
6. Leads V7, V8, V9 with posterior wall infaction.[46]

**ROLE OF 2D ECHO IN AMI:**

ASE has suggested 16-segment model or a 17-segment model with addition of the apical cap as a segment. The following score is applied to assess contractile function of left ventricle segments :

1 = normal (>40% thickening on systole)

2 = hypokinesis (10% to 40% thickening)

3 = akinesis (<10% thickening)

4 = dyskinesis

5 = aneurysm

This score is applied to each myocardial segment and total score is divided by the number of segments calculated.This is called as wall motion score index (WMSI).

ECHO is used to

1. To diagnose or rule out myocardial infarction
2. To assess infarct size
3. To assess myocardial viability
4. To detect infarct complications
5. For risk stratification
6. To assess infarct expansion
7. To assess myocardial segments

Persistent akinesis does not mean reperfusion failure. When the myocardium is akinetic but viable, low-dose dobutamine, contrast, or strain imaging echocardiography are useful to demonstrate its viability.[47] Regional strain or strain rate is also used as marker of acute ischemia.[48]

## CORRELATION OF ECG AND CAG IN MI :[49]

Following is the list of findings in ECG that correlate with angiographic findings of the concerned patients.

**RCA occlusion**:

ST depression in lead I & ST elevation in lead III greater than in lead II.

**Proximal :**

ST elevation more than 1mm with positive T wave in lead V4R.

**Distal:**

ST isoelectric with a positive T wave in lead V4R.

**LCX occlusion:**

ST elevation in lead II greater than lead III.

ST isoelectric or elevated in lead I.

ST isoelectric or depressed with negative T wave in V4R.

**LMCA occlusion :**

ST elevation in lead aVR

ST elevation in lead V1 (lower than that of lead aVR)

ST depression in leads II and aVF

ST depression in precordial leads to the left of V2.

**LAD occlusion:**

**Proximal to S1 and D1:**

ST elevation in leads aVR and aVL

ST depression in leads II III aVF

ST elevation in lead V1 (more than 2mm) and leads V2 to V4

ST isoelectric or depressed in leads V5 and V6

Acquired intra-Hisian block or RBBB

**Distal to S1 and proximal to D1**:

ST depression in lead I and aVL

ST depression in lead III (lead II is isoelectric)

ST elevation in leads V2 to V6 but not in lead V1

**Distal to D1 but proximal to S1:**

ST depression in lead aVL

ST elevation in inferior leads, highest in lead III

ST elevation in leads V1 to V4

**Distal LAD :**

ST depression in lead aVR

ST elevation in inferior leads, highest in lead II

ST elevation in leads V3 to V6.

## CLINICAL FEATURES OF COMBINED AWMI AND IWMI:

Patient of combined AWMI and IWMI has clinical features that are not different from other myocardial infarction patients. Patient has anginal chest pain which may be radiating to left arm, neck or back, dyspnea, diaphoresis, giddiness, easy fatiguability, palpitations, vomiting, epigastric discomfort, jaw pain and in some cases syncope.

Patient may have tachycardia or bradycardia and associated hypotension or hypertension. Some of the cases may present with or during the course may develop arrhythmias which are mostly of ventricular in origin.

Heart blocks can occur ranging from first degree to third degree complete AV block.

Some patients may develop Dressler's syndrome during the course in the hospital which has postural chest pain.

Anxiety of impending doom is an associated feature that to be treated by sedation as the patient warrants.

Major clinical outcomes like, Recurrent ischemia, Re-infarction, cardiogenic shock and even death can occur.

ECG showing ST elevation in anterior and inferior leads have coronary lesion in distal LAD, showing ST elevation in anterior leads but ST depression in inferior leads have lesion in proximal LAD and showing ST elevation in anterior leads but ST isoelectric in inferior leads have lesion in mid LAD[62].

An inferior AMI due to RCA occlusion typically involves only the basal and mid portion of the inferior wall and spares the apex.[63] Because anterior AMI caused by more distal occlusion of an LAD spares the basal and mid portion of the anterior wall but also involves the apical portion of the inferior wall, the net ST deviation is directed inferiorly. This causes the ST elevation in leads II, III, and aVF.

Alternatively, because an anterior AMI caused by an occlusion of the proximal LAD most often involves the superiorly situated base of the

LV, the ST segment is most often directed superiorly. This results in ST depression in inferior leads.

The ST elevation genuinely representing superior epicardial injury will be seen as ST depression in inferior leads, and as ST elevation in less superiorly oriented leads I and aVL.[62]

In echocardiogram, patients with anterior and inferior ST elevations most often have mild to moderate LV dysfunction as less area of myocardium is affected. Severe LV dysfunction is rare.

Coronary lesion causing anterior wall and inferior wall myocardial infarction usually arises from either distal LAD of from proximal RCA. The coronary lesions can be of any type of SCAI classification of coronary lesions and most often they are of type A lesions. Type C lesions are rare. Type B lesions occur less commonly.

# AIM

(1) TO EVALUATE THE CLINICAL AND ANGIOGRAPHIC SIGNIFICANCE OF COMBINED ANTERIOR AND INFERIOR ST-SEGMENT ELEVATION IN THE PRESENTING ECG IN PATIENTS WITH AMI,

(2) TO DETERMINE WHETHER THE AMI SIZE AS MEASURED BY ALDRICH SCORE AND ST ELEVATION IN THE LEADS $V_1$ TO $V_5$ MAY RELATE TO THE CORONARY ANGIOGRAPHIC LESION.

# MATERIALS AND METHODS

**Inclusion criteria:**

1. Age from 18-75 years
2. Acute coronary syndrome patients admitting in our ICCU
3. Patients presenting with AWMI & IWMI

**Exclusion criteria:**

1. Patients with previous history of myocardial infarction
2. Previous history of Left ventricular dysfunction
3. Previous history of Percutaneous coronary intervention
4. Previous history of coronary artery bypass graft
5. Patients with peripheral arterial disease
6. Patients with chronic kidney disease
7. Previous history of valve diseases or arrhythmias such as atrial fibrillation.

**Data collection technique and tools**

**Ethical issues:**

This study involves investigations, blood tests,medications, coronary angiograms, primary and rescue angioplasties, all patients and their relatives were explained the study design at the time of enrollment.

The following informationwas obtained at the time of admission:

Filling a proforma (a copy of which enclosed in appendix) for each patient which includes

**Detailed history:**

- Age of the patient
- Nature of symptoms and their duration
- An entire risk factor assessment (including diabetes, hypertension, dyslipidemia, smoking, family history, prior history of CAD).

**Thorough physical examination**

- Performed at the time of admission, or after stabilization as per the condition of patient as needed

**Blood sampling**

- At the time of admission and serially, if required were done

**Investigations**

- A 12 lead electrocardiogram was obtained. ECG using right sided leads was taken if necessary.

**During In-hospital course:**

1. Serial ECG's were taken; area involved, presence of conduction disturbances or arrhythmias were also noted
2. ECG score was calculated for the patients showing ST elevation in anterior and inferior leads (Aldrich score) which indicates the size of myocardial infarct in our study.[50] Aldrich score is calculated by multiplying 4.5 with the figure that comes from substracting 0.4 from the number of leads with ST elevation.[50]
3. A complete echocardiogram was done in all patients on admission and prior to discharge.
4. Complete blood investigations including blood counts, blood sugar, urea, creatinine, lipid profile, liver function test, CPK-MB, HBsAg, HCV, HIV and Chest X-Ray PA view taken at the time of admission.
5. Coronary angiogram was done in all patients during index hospitalisation or as a delayed strategy at a later date.

Echocardiography was performed after admission using Philips machineModel No: HD11XE 2011 USA Revision 2.0.5. All studies were performed and reviewed by staff cardiologists.

LV systolic function was estimated with a visually assessed ejection fraction and wall-motion score index. Complete patient characteristics, treatment details including Thrombolysis, Inotropic Therapy and coronary interventions findings were analysed.

All patients underwent coronary angiography within the first 5 days after admission, with a mean of 1.7 days between admission time and angiography. Coronary angiogram was done in SIEMENS Make 2000- Axiom Artis U Machine with Axiom Sensis Pressure Monitor. The arterial lesion was considered significant when it produced a reduction in lumen size of less than 70%.

Complications during in-hospital stay like LV failure, Arrhythmias, Re-MI, Mechanical Complications like VSR or MR, and Death were analysed.

**Analysis of the Results:**

The complete data collected in this study were compiled, analyzed and interpreted in relation to the objectives of this study.

**Statistical Analysis:**

Descriptive and inferential statistics were utilised to analyse the data of this study. The quantitative data are expressed as Mean ±

Standard Deviation. Qualitative data are expressed as frequency and percentage. The probability value (P value) of less than 0.05 has been considered as a significant value. Statistical analysis by using the SPSS software Version 16.0 and Independent t test. Non parametric test chi square was performed for categorical data.

Pearson chi square test has been used to compare the parameters including the In-hospital events.

# RESULTS AND DATA ANALYSIS

Here we are going to analyse the data collected from the 52 patients of our study. Following are the tables showing the analysed data in respect to the variables and their significance.

**Table-1**

**Age distribution of the study sample**

| **Age group (in Years)** | **N** | **%** |
|---|---|---|
| ≤ 40 | 2 | 3.80 |
| 41 – 50 | 15 | 28.80 |
| 51 - 60 | 25 | 48.10 |
| 61 – 70 | 10 | 19.20 |
| TOTAL | 52 | 100 |

Table 1 showing the age-wise distribution of patients.

Majority of incidence is among the patients in the age group of 51-60 that is 48.1%. Least number of patients are in the age of less than 40 years, that is 3.8%.Youngest patient was 37 years old and the oldest patient was 70 years old.

**TABLE-2**

**Age distribution by sex**

| Age group (in Years) | MALE | | FEMALE | |
|---|---|---|---|---|
| | N | % | N | % |
| ≤ 40 | 2 | 4.80 | 0 | 0 |
| 41 – 50 | 13 | 31.00 | 2 | 20.00 |
| 51 - 60 | 22 | 52.40 | 3 | 30.00 |
| 61 – 70 | 5 | 11.90 | 5 | 50.00 |
| **TOTAL** | **42** | **100** | **10** | **100** |
| **Mean** | 52.90±7.20 | | 58.90±7.59 | |
| **Significant** | t=2.34 df=50 p=0.02 Significant | | | |

This table shows sex-wise distribution of patients in our study.

Mean age of male patients is 52.90±7.20

Mean age of female patients is 58.90±7.59

Difference between the male and female age distribution in this study is significant.

**Table-3**

**Diabetic status of the study subjects**

| Diabetic Status | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | N | % | N | % | N | % |
| Present | 18 | 42.90 | 8 | 80.00 | 26 | 50.00 |
| Absent | 24 | 57.10 | 2 | 20.00 | 26 | 50.00 |
| Total | 42 | 100 | 10 | 100 | 52 | 100 |
| significant | Chi square = 4.46 df = 1 p = 0.04 Significant | | | | | |

This table shows the distribution of diabetes mellitus among our study patients.

Diabetes mellitus is present in 42.90% of male patients and in 80% of female patients. This finding is statistically significant as the p value for this is 0.04.

**Table – 4**

**Hypertension status**

| Hypertension | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | N | % | N | % | N | % |
| Present | 25 | 59.50 | 4 | 40.00 | 29 | 55.80 |
| Absent | 17 | 40.50 | 6 | 60.00 | 23 | 44.20 |
| Total | 42 | 100 | 10 | 100 | 52 | 100 |
| significant | Chi square = 1.25 df = 1 p =0.25 . Not Significant | | | | | |

This table depicts the distribution of systemic hypertension among our study patients which shows the presence of hypertension as 55.8% and normotensives as 44.2% and the sex-wise distribution of this, in males as 59.5% and in females as 40%. The p value for this is 0.25 which shows the statistical insignificance.

**Table – 5**

**Smoking Status**

| Smoking | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | N | % | N | % | N | % |
| Present | 31 | 73.80 | 0 | 0 | 31 | 59.60 |
| Absent | 11 | 26.20 | 10 | 100 | 21 | 40.40 |
| Total | 42 | 100 | 10 | 100 | 52 | 100 |
| significant | Chi square = 18.28 df = 1 p =0.000 Significant | | | | | |

This table shows the distribution of smoking habit among our study patients which shows the presence of the habit as 59.6% and the non smokers as 40.4%. The sex-wise distribution of smoking habit is 73.8% in males and is 0% in females which shows the smoking habit is not much prevalent among females in Indian culture. The p value for these observations is 0.000 and is statistically much significant.

**Table - 6**

**Dyslipidemia**

| Smoking | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | N | % | N | % | N | % |
| Present | 15 | 35.70 | 5 | 50.00 | 20 | 38.50 |
| Absent | 27 | 64.30 | 5 | 50.00 | 32 | 61.50 |
| Total | 42 | 100 | 10 | 100 | 52 | 100 |
| Significant | Chi square = 0.70 df = 1 p =0.40 Not Significant | | | | | |

This table shows the distribution of dyslipidemia among our study patients. This shows presence of dyslipidemia in 38.5% of patients and its absence in 61.5% of patients and sex distribution of dyslipidemia here is 35.7% in males and 50% in females. The p value for these data is 0.40 which is of statistical insignificance.

**Table-7 Height (CM)**

| Height | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | **Mean** | **Sd** | **Mean** | **Sd** | **Mean** | **sd** |
| | 167.21 | 3.63 | 158.50 | 5.32 | 165.54 | 5.26 |
| Significant | t=6.21 df=50 p=0.000 Significant | | | | | |

| Height (cm) | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | **N** | **%** | **N** | **%** | **N** | **%** |
| 150-160 | 01 | 2.40 | 6 | 60.00 | 7 | 13.50 |
| 160-170 | 36 | 85.70 | 4 | 40.00 | 40 | 76.90 |
| 170-180 | 5 | 11.90 | 0 | 0 | 5 | 9.60 |
| TOTAL | 42 | 100 | 10 | 100 | 52 | 100 |
| Significant | Chi square =23.30 df=20 p=0.000 Significant | | | | | |

This table shows the distribution of height among our study patients with a mean height of 165.54cm and a standard deviation of 5.26. Males have a mean height of 167.21cm and females have a value of 158.50cm with a standard deviation of 3.63 and 5.32 respectively. The p value for these observations is 0.000 and is statistically significant.

**Table-8 Weight (kg)**

| Weight | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | **Mean** | **Sd** | **Mean** | **Sd** | **Mean** | **Sd** |
| | 77.43 | 6.44 | 71.80 | 5.09 | 76.35 | 6.55 |
| Significant | t=2.57 df=50 p=0.01 Significant | | | | | |

| Weight (Kg) | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | **N** | **%** | **N** | **%** | **N** | **%** |
| 60-70 | 7 | 16.70 | 5 | 50.00 | 12 | 23.10 |
| 70-80 | 19 | 45.20 | 4 | 40.00 | 23 | 44.20 |
| 80-90 | 16 | 38.10 | 1 | 10.00 | 17 | 32.70 |
| TOTAL | 42 | 100 | 10 | 100 | 52 | 100 |
| Significant | Chi square =5.89 df=2 p=0.05 Significant | | | | | |

This table shows the distribution of weight among our study patients with a mean weight of 76.35 and a standard deviation of 6.55. Males have a mean weight of 77.43 and females have a value of 71.80 with a standard deviation of 6.44 and 5.09 respectively. The p value for these observations is 0.01 and is statistically significant.

**Table-9** **BMI**

| **BMI** | **Male** | | **Female** | | **Total** | |
|---|---|---|---|---|---|---|
| | **Mean** | **Sd** | **Mean** | **Sd** | **Mean** | **sd** |
| | 27.67 | 1.91 | 28.56 | 1.71 | 27.84 | 1.89 |
| Significant | t=1.35 df=50 p=0.18 Not Significant | | | | | |

| **BMI** | **Male** | | **Female** | | **Total** | |
|---|---|---|---|---|---|---|
| | **N** | **%** | **N** | **%** | **N** | **%** |
| NOT Obese | 36 | 85.70 | 8 | 80.00 | 44 | 84.60 |
| Obese | 6 | 14.30 | 2 | 20.00 | 8 | 15.40 |
| TOTAL | 42 | 100 | 10 | 100 | 52 | 100 |
| Significant | Chi square =0.20 df=1 p=0.65 Not Significant | | | | | |

This table shows the distribution of body mass index in our study patients and it shows obesity in 15.4% of patients and 84.6% of our study patients are not obese. The mean body mass index is 27.84 with SD of 1.89. For males, the mean BMI is 27.67 with an SD of 1.91. For females, the mean BMI is 28.56 with an SD of 1.71. The p value for these observations is 0.65 which is not of much statistical significance.

**Table-10**

**KILLIP CLASS**

| KILLIP | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | N | % | N | % | N | % |
| Class-I | 20 | 47.60 | 7 | 70.00 | 27 | 51.90 |
| Class-II | 16 | 38.10 | 2 | 20.00 | 18 | 34.60 |
| Class-III | 4 | 9.50 | 1 | 10.00 | 5 | 9.60 |
| Class-IV | 2 | 4.80 | 0 | 0 | 2 | 3.80 |
| TOTAL | 42 | 100 | 10 | 100 | 52 | 100 |
| Significant | Chi square =2.02 df=3 p=0.56 Not Significant | | | | | |

The distribution of killip class on presentation of our study patients has been shown here. Majority of our study patients presented with killip class I, that is 51.9%, patients presented with killip class II are 34.6%, patients admitted with killip class III are 9.6% and those who presented with killip class IV are only 3.8% and the p value for these findings is 0.56 which is statistically not significant.

**TABLE-11**

**INOTROPIC USE**

| | **Male** | | **Female** | | **Total** | |
|---|---|---|---|---|---|---|
| | **N** | **%** | **N** | **%** | **N** | **%** |
| NO | 31 | 73.80 | 9 | 90.00 | 40 | 76.90 |
| YES | 11 | 26.20 | 1 | 10.00 | 12 | 23.10 |
| TOTAL | 42 | 100 | 10 | 100 | 52 | 100 |
| Significant | Chi square =1.19 df=1 p=0.28 Not Significant | | | | | |

The use of inotropic agents in our study patients are showed in this table which says yes to 23.1% of our patients and no to 76.9% of our study patients. Inotropic drugs are used in 26.2% of male patients and in 10% of female patients and the p value for these observations is 0.28 which is not statistically significant.

**Table-12**

**THROMBOLYSIS**

| | **Male** | | **Female** | | **Total** | |
|---|---|---|---|---|---|---|
| | **N** | **%** | **N** | **%** | **N** | **%** |
| NOT LYSED | 2 | 4.80 | 1 | 10.00 | 3 | 5.80 |
| LYSED | 40 | 95.20 | 9 | 90.00 | 49 | 94.20 |
| TOTAL | 42 | 100 | 10 | 100 | 52 | 100 |
| Significant | Chi square =0.41 df=1 p=0.52 Not Significant | | | | | |

This table shows thrombolysis in our study patients showing 94.2% patients are lysed and 5.8% patients are not lysed due to the late presentation. Among males, 95.2% are lysed and 4.8% are not lysed. Among females, 90% are lysed and 10% are not lysed. The p value for these observations is 0.52 which is statistically not significant.

**TABLE-13**

**ARRYTHMIAS**

| | **Male** | | **Female** | | **Total** | |
|---|---|---|---|---|---|---|
| | **N** | **%** | **N** | **%** | **N** | **%** |
| NO | 29 | 69.00 | 8 | 80.00 | 37 | 71.20 |
| VENTRICULAR | 13 | 31.00 | 2 | 20.00 | 15 | 28.80 |
| TOTAL | 42 | 100 | 10 | 100 | 52 | 100 |
| Significant | Chi square =0.47 df=1 p=0.49<br>Not Significant | | | | | |

The incidence of arrhythmias in our study patients during the hospital course which are always ventricular in origin and has an incidence rate of 28.8% with a distribution of 31% in male patients and 20% in female patients with p value of 0.49 which is statistically not significant.

**TABLE-14**

**AV BLOCK**

| | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | N | % | N | % | N | % |
| NO | 40 | 95.20 | 10 | 100 | 50 | 96.20 |
| YES | 2 | 4.80 | 0 | 0 | 2 | 3.80 |
| TOTAL | 42 | 100 | 10 | 100 | 52 | 100 |
| Significant | Chi square =0.50 df=1 p=0.48 Not Significant | | | | | |

The incidence of AV block in our study patients has been shown here which is 3.8% totally. Among male patients the incidence of AV block is 4.8% and among females AV block is 0% in our study. The p value for these findings is 0.48 which is not statistically significant.

**TABLE-15 EF %**

| | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | **Mean** | **Sd** | **Mean** | **Sd** | **Mean** | **Sd** |
| | 39.38 | 4.74 | 40.10 | 3.76 | 39.52 | 4.55 |
| Significant | t=0.45 df=50 p=0.66 Not Significant | | | | | |

| | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | **N** | **%** | **N** | **%** | **N** | **%** |
| 30-35 | 9 | 21.40 | 1 | 10.00 | 10 | 19.20 |
| 35-40 | 12 | 28.60 | 4 | 40.00 | 16 | 30.80 |
| 40-45 | 19 | 45.20 | 5 | 50.00 | 24 | 46.20 |
| 45-50 | 2 | 4.80 | 0 | 0 | 2 | 3.80 |
| TOTAL | 42 | 100 | 10 | 100 | 52 | 100 |
| Significant | Chi square =1.41 df=3 p=0.70 Not Significant | | | | | |

The left ventricular ejection fraction values are assessed in our patients and it shows a mean value of 39.52 with a standard deviation of 4.55 and among males the mean EF is 39.38 with an SD of 4.74 and among females the mean EF is 40.10 with an SD of 3.76 and the p value for these values is 0.66 which is statistically not significant.

**Table-16 ECG SCORE (ALDRICH)**

| | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | **Mean** | **Sd** | **Mean** | **Sd** | **Mean** | **Sd** |
| | 18.77 | 3.17 | 18.00 | 2.32 | 18.62 | 3.02 |
| Significant | t=0.0.72 df=50 p=0.47 Not Significant | | | | | |

| | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | **N** | **%** | **N** | **%** | **N** | **%** |
| 16.2 | 23 | 54.80 | 6 | 60.00 | 29 | 55.80 |
| 20.7 | 14 | 33.30 | 4 | 40.00 | 18 | 34.60 |
| 25.2 | 5 | | 0 | | 5 | 9.60 |
| TOTAL | 42 | 100 | 10 | 100 | 52 | 100 |
| Significant | Chi square =1.33 df=2 p=0.51 Not Significant | | | | | |

The ECG score (Aldrich score) which indicates the size of myocardial infarct[50] and the mean value for this is 18.62 with an SD of 3.02 and in male patients the mean ECG score is 18.77 with an SD of 3.17 and in female patients the mean ECG score is 18.00 with an SD of 2.32 which has the p value of 0.47 that is not statistically significant. The ECG score of 16.2 is found in 55.8% of patients and score of 20.7 is found in 34.6% of patients and a score of 25.2 is found only in 9.6% of patients.

**TABLE-17 LMCA LESIONS**

| | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | **N** | **%** | **N** | **%** | **N** | **%** |
| Normal | 40 | 95.20 | 10 | 100 | 50 | 96.20 |
| Distal30% | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| Distal70% | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| TOTAL | 42 | 100 | 10 | 100 | 52 | 100 |
| Significant | Chi square =0.50 df=2 p=0.78 Not Significant | | | | | |

The LMCA lesion is analysed in our study patients which shows absence of LMCA lesion in 96.2% of patients and distal LMCA 30% lesion in 1.9% of cases and distal 70% lesion of LMCA is found in 1.9% of patients and the p value for these observations is 0.78, that is not statistically significant.

**TABLE 18 LAD LESIONS**

| | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | **N** | **%** | **N** | **%** | **N** | **%** |
| NO Lesion | 2 | 4.80 | 2 | 20.0 | 4 | 7.60 |
| Distal 50% | 2 | 4.80 | 0 | 0 | 2 | 3.80 |
| Distal 70 % | 2 | 4.80 | 0 | 0 | 2 | 3.80 |
| Distal 90% | 11 | 26.10 | 6 | 60 | 17 | 33.30 |
| Distal 99% | 4 | 9.60 | 0 | 0 | 4 | 7.70 |
| MID 50 %, distal 70% | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| Mid 50% | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| Mid 70% | 2 | 4.80 | 1 | 10.00 | 3 | 5.70 |
| Mid 90% | 1 | 2.40 | 0 | 10.00 | 1 | 1.90 |
| Mid &distal 70% | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| Mid irregularities | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| Mid 100% | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| Ostial 50% | 0 | 0 | 1 | 10.00 | 1 | 1.90 |
| Proxmal 70% | 2 | 4.80 | 0 | 0 | 2 | 3.80 |
| Proximal 90% | 4 | 9.50 | 0 | 0 | 4 | 7.70 |
| Proximal 99% | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| Proximal 100% | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| Prox 70% &mid 100% | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| TOTAL | 42 | 100 | 10 | 100 | 52 | 100 |
| Significant | Chi squre=16.34 df=20 p=0.70 Not significant | | | | | |

This table shows the distribution of lesions in proximal, mid, distal LAD with various amount of severity of lesions.

These observations show that majority of cases have distal LAD lesion, that is 48.6% when more than 70% stenosis is taken into account, even though they are having significant or insignificant lesions in other coronary arteries.

About 7.6% of patients were having no lesion in LAD. Proximal LAD lesion with more than 70% stenosis is seen in 17.2% of our study patients and ostial LAD lesion is seen in 1.9% of patients and mid LAD lesion of more than 70% is seen in 11.4% of our study patients.

The p value of these observations is 0.70 which is statistically not significant.

**TABLE-19 LCX LESIONS**

| | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | **N** | **%** | **N** | **%** | **N** | **%** |
| No | 31 | 73.80 | 8 | 80.0 | 39 | 75.00 |
| Distal50% | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| Distal70% | 2 | 4.80 | 0 | 0 | 2 | 3.80 |
| Distal90% | 0 | 0 | 1 | 10.00 | 1 | 1.90 |
| Distal99% | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| Prox 50 % | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| Prox 70 % | 6 | 14.30 | 0 | 0 | 6 | 11.50 |
| Ostial70% | 0 | 0 | 1 | 10.00 | 1 | 1.90 |
| TOTAL | 42 | 100 | 10 | 100 | 52 | 100 |
| Significant | Chi square =11.06 df=7 p=0.14 Not Significant | | | | | |

This table shows the distribution of lesions in left circumflex coronary artery which shows 7.6% of patients were having more than 70% distal LCX lesion, 11.5% havemore than 70% proximal LCX lesion and 75% of patients were not having any LCX lesions. The p value for these observations is 0.14 and this is not statistically significant.

**Table-20 RCA LESIONS**

| | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | N | % | N | % | N | % |
| Normal | 23 | 54.80 | 6 | 60.00 | 29 | 55.80 |
| Distal 100 | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| Mid 80 | 0 | 0 | 1 | 10.00 | 1 | 1.90 |
| Mid 90 | 7 | 16.70 | 1 | 10.00 | 8 | 15.40 |
| Ostial 50 | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| Prox 70 | 3 | 7.10 | 0 | 0 | 3 | 5.80 |
| Prox 100 | 0 | 0 | 1 | 10.00 | 1 | 1.90 |
| Prox 40 | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| Prox 50 | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| Prox70 | 1 | 2.40 | 1 | 10.00 | 2 | 3.80 |
| Prox 70 &MID70% | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| Prox 99 | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| Prox 90 | 2 | 4.80 | 0 | 0 | 2 | 3.80 |
| TOTAL | 42 | 100 | 10 | 100 | 52 | 100 |
| Significant | Chi square =12.51 df=12 p=0.41 Not Significant | | | | | |

This table shows the distribution of coronary lesions in right coronary artery in our study patients in which 55.8% of patients have no lesion in RCA. Distal RCA with 100% lesion is seen in 1.9% of patients. Mid RCA lesion with more than 70% stenosis is seen in 19.2% of patients. Proximal RCA lesion with more than 70% stenosis is seen in 19.1% of patients. The other patients were having less than 70% lesions in RCA and the p value for these observations is 0.41 which is not significant statistically.

**Table 21**

**SCAI Lesions Type**

| **Type** | **NUMBER** | **PERCENTAGE** |
|---|---|---|
| **Type A** | 43 | 83 |
| **Type B** | 7 | 13 |
| **Type C** | 2 | 4 |
| **Total** | 52 | 100 |

The SCAI type of coronary lesions are shown in this table.

Patients with Type A coronary lesion were about 83%. Patients with Type B coronary lesion were about 13%. Patients with Type C coronary lesion were about 4%. So, the most common type of coronary lesion in our study is Type A.

**Table-22 PCI**

| | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | **N** | **%** | **N** | **%** | **N** | **%** |
| No | 40 | 95.20 | 9 | 90.00 | 49 | 94.20 |
| Done | 1 | 2.40 | 1 | 10.00 | 2 | 3.80 |
| Primary PCI Done | 1 | 2.40 | 0 | 0 | 1 | 1.90 |
| TOTAL | 42 | 100 | 10 | 100 | 52 | 100 |
| Significant | Chi square =1.48 df=2 p=0.48 Not Significant | | | | | |

This table contributes to the observations on patients of our study with PCI done and not done. In 3.8% of patients PCI was done and in 94.2% of patients PCI was not done and these observations have p value of 0.48, that is statistically not significant.

**Table-23 Death**

| | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | **N** | **%** | **N** | **%** | **N** | **%** |
| No | 40 | 95.20 | 10 | 100 | 50 | 96.20 |
| Death | 2 | 4.80 | 0 | 0 | 2 | 3.80 |
| TOTAL | 42 | 100 | 10 | 100 | 52 | 100 |
| Significant | Chi square =0.50 df=1 p=0.48 Not Significant | | | | | |

This table is showing the occurrence of death in our study patients which is 3.8% and among males, it is 4.8% and among females, it is 0% with p value of 0.48 which is statistically not significant.

**Table 24 Comparison of Angiogram lesions**

| Coronary artery | **LMCA** | | **LAD** | | **LCX** | | **RCA** | |
|---|---|---|---|---|---|---|---|---|
| | Number | % | Number | % | Number | % | Number | % |
| Negative | 50 | 96.2 | 4 | 7.6 | 39 | 75.3 | 29 | 56.3 |
| Positive | 2 | 3.8 | 48 | 92.4 | 13 | 24.7 | 23 | 43.7 |
| Total | 52 | 100 | 52 | 100 | 52 | 100 | 52 | 100 |

This table is showing the distribution of angiographic lesions in coronary arteries. Among them, LAD had the highest lesion rate of 92.4%, RCA being the second highest lesion holder by having a rate of 43.7%, LCX had lesion rate of 24.7% and the least rate of lesion occurrence was with LMCA, that is 3.8%.

# DISCUSSION

Here we are going to discuss the data that were obtained during our study and subsequently analysed statistically.Patients who were presented with acute myocardial infarction with the ECG changes of combined ST elevation in anterior and inferior leads, were examined and the obtained data were analysed statistically. The variables were observed for any peculiar significance in such patients and the findings are now discussed.

In this study 52 patients were studied and data were analysed. Among the 52 patients, 42 were male and 10 were female, reporting a probable lower incidence of combined ST elevation in anterior and inferior leads during acute MI in females.

Majority of our studypatients fall in the age group of 51 to 60 years, that is 48.1%. Mean age for the male patients is around 52 and mean age for female patients is around 58 which becomes a statistically significant one, suggesting the occurrence of combined AWMI and IWMI later in life in females.

Prevalence of diabetes mellitus in our study patients is more in females than in males,that is 80% versus 43%, suggesting a major causative factor in women and suggesting the role of other causative factors also in males.

Systemic hypertension is prevalent in about 56% of our patients and about 44% of our patients were normotensive. About 59% of male patients and 40% of female patients are hypertensive suggesting a statistically insignificant causative role in our study patients.

Smoking plays a statistically significant major causative role in our study patients, showing 74% of male patients were smokers and 26% of them were non smokers. Being in Indian culture, all of our female patients were non smokers.

Dyslipidemia is present 38% of our patients and absent in 62% of our patients. Male patients had lesser incidence of dyslipidemia than female patients, that is 36% versus 50% suggesting an important role in female patients.

Body mass index analysis in our study patients shows obesity in about 15% of patients and about 85% of our study patients are not obese. The mean body mass index is 27.84 with SD of 1.89. For males, the mean BMI is 27.67 with an SD of 1.91. For females, the mean BMI is 28.56 with an SD of 1.71. The p value for these observations is 0.65 which is not of much statistical significance.

Majority of AWMI and IWMI patients presented in Killip class I, that is 52% and the least presented Killip class is IV, which is 3.8%.

Inotropic drugs were used in 23% of our patients and not needed in 77% of our patients suggesting a not much of need in AWMI and IWMI cases.

In our study, 94% of our cases were thrombolysed with streptokinase and about 6% of our cases were not thrombolysed because of their late presentation.

In this study, 29% of patients had ventricular arrhythmias and 71% of patients did not have any arrhythmic episode during their hospital stay. All the arrhythmias were of ventricular in origin and no atrial or supraventricular arrhythmias were found.

The incidence of AV block in our study patients is only 4% and it was 2:1 second degree type II AV block in both the patients and 96% were free from AV block features. The AV block patients were recovered from their AV block after treatment during their hospital stay.

In our study, about 46% of patients had an ejection fraction of 40-45% and about 31% of patients had ejection fraction of 35-40%. About 4% of patients had an ejection fraction of 45-50% and about 19% had an ejection fraction of 30-35%. These observations show majority of our patients were in the range of moderate LV dysfunction.

The Aldrich ECG scores obtained in our study patients, which indicate the size of myocardial infarction[50], show that majority of them, that is about 60%, were with the ECG score of 16.2which is the lowest score and the patients with higher ECG score were around 9% showing that most of the AWMI and IWMI patients have lower ECG scores contrary to what would be expected.

The median sum of ST elevation in leads II, III, and aVF was much higher in patients with RCA lesion when compared to LAD lesion (4 mmvs2mm, $P = 0.05$). The median sum of ST elevation in lead $V_3$ was much higher in patients with LAD lesions when compared with RCA lesions(4 mm vs1 mm, $P = 0.04$). Progression of ST elevation from $V_1$ to $V_3$ was calculated as ratio of ST elevation in $V_3$ to $V_1$ and the ratio was higher in patients with LAD lesion when compared to patients havinglesion in RCA (ratio of ST elevation $V_3$ to $V_1$ is 4 : 1, $P = 0.04$). Thus, larger ST elevation in leads II, III, and aVF, and absence of progression of ST elevation from $V_1$ to $V_3$ identifies RCA lesion from LAD lesion in patients with combined anterior and inferior ST elevation.

Lead $V_1$ is as much a “posterior” as an “anterior” lead, and lead aVF is as much a “superior” as an “inferior” lead.[61]Anterior and posterior deviation of the ST segment in the horizontal plane are reflected as $V_1$ to

$V_3$ ST elevation and depression respectively, and so that they can be appropriately called as anterior and posterior ST elevationrespectively.[61] Similarly, inferior and superior deviation of the ST segment in the frontal plane are reflected as II, III, and aVF ST elevation and depression respectively, and so that they can be appropriately called as inferior and superior ST elevation.[61]Anterior ST elevation with inferior ST depression could be better termed “anterior and superior ST elevation”.[61]

**Description and rationale for new terminology to describe ST deviation in precordial and limb leads[61]**

| Description of terminology | Appropriate terminology |
|---|---|
| ST elevation in leads $V_1$–$V_4$ | Anterior ST elevation |
| ST depression in leads $V_1$–$V_4$ | Posterior ST elevation |
| ST elevation in leads II, III, aVF | Inferior ST elevation |
| ST depression in leads II, III, aVF | Superior ST elevation |
| ST elevation in leads $V_1$–$V_4$ and II, III, aVF | Anterior and inferior ST elevation |
| ST elevation in leads $V_1$–$V_4$ and ST depression in leads II, III, aVF | Anterior and superior ST elevation |

In Acute MI due to LAD lesion, inferior ST elevation necessitates 2 conditions: (1) sparing of the basal anterior wall, causing no superiorly oriented ST-segment deviation and so, no reciprocal inferior ST depression, (2) involvement of LV inferior wall apical segment. Sapin et al described these findings in 42 patients with anterior AMI.[51] In RVMI, the ST elevation in $V_1$ to $V_4$ is opposed by posterior injury current that isoften associated with the MI.[54] Alternatively, acute RV injury and dilatation as a result of proximal RCA occlusion may lead to larger part of RV free wall directed anteriorly and ST elevation may therefore be seen in the leads $V_1$ to $V_4$.[52,53]

According to Geft et al, ST elevation, in RVMI, was highest in $V_1$ to $V_2$ and decreased towards $V_5$.[55] In contrast, in AWMI due to LAD occlusion, the ST elevation was least in $V_1$ and progressively increased towards $V_5$. Our data correlate with these observations and indicate that in patients with both anterior and inferior ST-elevation AMI on ECG, ST elevation in $V_1 \geq V_3$ and absence of progression of ST elevation from lead $V_1$ to $V_3$ can differentiate RCA as the IRA from LAD.

LAD extending around apex for some distance to supply the apical part of inferior wall is called‘wrap around LAD’.[56] TheIWMI due to RCA occlusion involves basal, mid portions of the inferior wall

and spares the apex.[57]As AWMI due to distal LAD spares the basal, mid portions of the anterior wall but involving the apical part of the inferior wall, the net ST deviation is directed inferiorly. This results in ST elevation in leads II, III, and aVF.

Wilkins et al studied the effectiveness of the Aldrich score and demonstrated that the number of leads with ST elevation was one of the strongest predictors ofthe AMI size.[58] Mauri et al studied 8731 patients with first Q-wave AMI enrolled in the GISSI- I trial, and showed a direct correlation between the number of leads with ST elevation and mortality.[59]

As far as the coronary artery lesions as assessed by coronary angiogram were concerned, LMCA lesion is not a significant one in cases of AWMI and IWMI. In our study, LMCA was normal in 96.2% of cases and only 3.8% of cases had lesion in LMCA, that too in distal LMCA of which one is 30% lesion.

As far as the LAD lesions were concerned, only 7.6% of patients had normal LAD. Significant proximal LAD lesions, that is more than 70%, were present in 17.2% of patients and more than 70% lesions in mid LAD were present in 11.4% of patients. The most important and astonishing finding is involvement of distal LAD. Distal LAD lesions of

more than 70% stenosis were seen in 48.6% of patients and all of them had Type III LAD (wrap around LAD). Thus, distal LAD becomes the important culprit segmentof LAD artery involving majority of patients with AWMI and IWMI in our study which correlateswell with the other studies done by Saihari Sadanandan et al.[62]

As far as the LCX lesions were concerned in our study, 75% of patients were having normal LCX and 7.6% of patients were having more than 70% distal LCX lesion, 11.5% have more than 70% proximal LCX lesion. These findings suggest LCX is not a major culprit coronary artery in patients with AWMI and IWMI.

As far as the RCA lesions were concerned in our study in which 55.8% of patients have no lesion in RCA. Distal RCA with 100% lesion is seen in 1.9% of patients. Mid RCA lesion with more than 70% stenosis is seen in 19.2% of patients. Proximal RCA lesion with more than 70% stenosis is seen in 19.1% of patients.

When Proximal and mid RCA are combined, their contribution to our study population comes around 38% which is of some considerable significance. Only 1.9% of patients had both proximal and mid RCA lesion with more than 70% stenosis. So, proximal and mid RCA when

combined, occupies the second place among the culprit segments of coronary arteries in causing AWMI and IWMI in our study patients.

In our study, 57.7% of patients had SVD, 25% of patients had DVD and 17.3% of patients had TVD in their coronary angiograms.

In our study, 3.8% of patients had undergone PCI and in 94.2% of patients PCI was not done and these observations also indicate Primary PCI in 1.9% of patients.

Death occurred in our study patientsof 3.8%who presented late with time window more than 24 hours and in Killip class IV. Among males, death rate is 4.8% andamong females, it is zero percentage.

# STUDY LIMITATIONS

1. Study was conducted in single centre and tested sample size was small. Both of these can cause selection bias.
2. Long term follow up of patients is not available.
3. Coronary angiographic assessment is based on luminal assessment and lacks plaque visualisation which requires Intravascular ultrasound (IVUS).

# CONCLUSION

1. In cases presenting with combined anterior and inferior ST segment elevation on ECG during Acute MI, males were more affected than females.

2. Majority of patients presented with Killip class I and Moderate LV Dysfunction.

3. Majority of patients had lower Aldrich score predicting lesser area of myocardium is affected.

4. Majority of patients had single vessel disease in coronary angiogram.

5. The coronary angiographic lesion causing combined anterior and inferior ST segment elevation was most commonly Distal LAD, followed by Proximal RCA.

6. ST elevation more inlead V1 than in lead V3 indicates Proximal RCA lesion in the presence of ST elevation in inferior leads.

7. ST elevation more in leadV3 than in lead V1 indicates Distal LAD lesion in the presence of ST elevation in inferior leads.

8. Patients with lower Aldrich scores have more Distal LAD lesions than those with the higher scores who had more Proximal LAD lesions than the distal ones.

9. In the presence of ST elevation in inferior leads, progressive increase in ST elevation from leads V1 to V5 indicates lesion in Distal LAD, and progressive decrease in ST segment elevation from leads V1 to V5 indicates lesion in Proximal RCA, both of which are important culprits in our study.

10. SCAI type of coronary lesions predominating in our study is type A lesion with 83%, followed by type B lesion with 13% and the least by type C lesion with 4%.

11. Further studies are needed in the future with large group of patients to further validate these findings.

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# INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Clinical and Angiographic characteristics of patients with combined anterior and inferior segment elevation on the initial electrocardiogram during acute myocardial infarction

Principal Investigator : Dr.A. Srinivasan

Designation : PG in D.M (Cardiology)

Department : Department of Cardiology
Government Stanley Medical College,
Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 07.02.2013 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
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1 INTRODUCTION Myocardial infarction is the common disease nowadays and ST elevation myocardial infarction is important one among the ACS (Acute Coronary Syndrome). It is quite rare a situation when patient presents with simultaneous ST elevation in both anterior and inferior leads during acute myocardial infarction. In this thesis, we are going to see the various implications of this rare presentation in myocardial involvement as assessed by electrocardiogram and distribution of lesions in coronary arteries as assessed by coronary angiogram. The life- saving and crucial use of reperfusion therapy for acute myocardial infarction (AMI)1 makes it important to find out the location of the...

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# PROFORMA

**Name:** **Age:**

**Sex:** **Occupation:**

**Address:**

**Presenting Complaints:**

**Diagnosis:** AWMI
IWMI

**Risk Factors:** Male Gender:
Smoker:
Hypertension:
Diabetes:
Obesity:
Hyperlipidemia:
Family History of CAD:

**In Hospital Therapy:** Thrombolysis:
Inotropic Therapy:

**General examination :**

HT: WT: BMI: BSA:

PR: BP:

**Systemicexamination** :

CVS: RS:

**Killip Class On Admission:**

**Investigations:**

**Complete Blood Count:**

Urea: Creatinine:

Electrolytes: Lipid profile:

CPK/MB:

**ECG:**

Aldrich score:

ST elevation in V1 to V6: ST elevation in II III VF**:**

**Chest X-Ray PA View:**

**ECHO:**

LVEDD:

LVESD:

LVEF, %:

WMSI:

DiastolicFunction:

**Coronaryangiographicfindings :**

LMCA :

LAD :

LCX :

RCA :

Ramus :

**PCI :**

**CABG :**

**In Hospital Events:**

Arrhythmias:

AV Block:

LV Dysfunction:

Mechanical Complication: VSR / MR

Re-MI:

Death :

# CONSENT FORM

I agree to participate in the study titled – '**Clinical and angiographic characteristics of patients with combined anterior and inferior ST-segment elevation on the initial electrocardiogram during acute myocardial infarction'.**

I confirm that I have been told about this study in my mother tongue and have had the opportunity to ask question.

I understand that my participation is voluntary and I may refuse to participate at any time without giving any reason and without affecting my benefits.

I agree not to restrict the use of any data or results that arise from the study.

I agree to undergo the necessary investigation which is part of the study.

Name of the participant:

Signature / thumb impression:

Investigator:

Witness:

# சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு

**இருதய மின்னலை வரைவுகளில் இருதய வெளிச்சுவர் மற்றும் உட்சுவர் மாரடைப்பு உள்ள நோயாளிகளில் மருத்துவ மற்றும் இருதய உட்புகுத்து ஆய்வு காரணிகளின் ஒப்பீட்டு ஆய்வு,**

ஆராய்ச்சி நிலையம் : அரசு ஸ்டான்லி மருத்துவமனை
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பங்கு பெறும் நோயாளியின் பெயர் : வயது :

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நோயாளியின் விலாசம்

நோயாளி இதனை ( ) குறிக்கவும்.

மேலே குறிப்பிடப்பட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும். அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது. [ ]

நான் என்னை இவ்வாய்வில் தன்னிச்சையாகதான் பங்கேற்க அனுமதிக்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் என்னை இவ்வாய்வில் இருந்து விலக்கி கொள்ளலாம் என்றும் அறிந்து கொண்டேன். [ ]

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். என்னை ஆய்வில் இருந்து விலக்கிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். [ ]

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். [ ]

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய்க்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணிக்கு தெரிவிப்பேன் என உறுதி அளிக்கிறேன். [ ]

நோயாளியின் கையொப்பம் ..... இடம் .....தேதி

கட்டைவிரல் ரேகை (இந்த படிவம் படித்து காட்டப்பட்டு புரிந்து கைரேகை அளிக்கின்றேன்)

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

.....

ஆய்வாளரின் கையொப்பம் ..... இடம் ..... தேதி

ஆய்வாளரின் பெயர்.....

| NAME | AGE | SEX | DM | HT | Smoking | Dyslipidemia | HEIGHT | Weight | BMI | DIAGNOSIS | KILLIP | CPK-MB | Inotropic | Thrombolysis | Arrythmias | AV Block | EF% | ECG Score | LMCA | LAD | LCX | RCA | Ramus | typeA | typeB | typeC | PCI | Death |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Suresh kumar | 41 | m | | Present | Present | Present | 152 | 64 | 27.7 | awmi+iwmi | class I | elevated | | lysed | | | 45% | 21 | | 90% astride D1 origin | | | | yes | | | | |
| Thanu | 67 | M | Present | | Present | | 165 | 72 | 26.4 | awmi+iwmi | class I | elevated | | lysed | | | 48% | 16 | | 99%distal | | ostial50% | | yes | | | | |
| Ali akbar | 45 | M | Present | Present | | Present | 161 | 73 | 28.1 | awmi+iwmi | class I | elevated | | lysed | | | 42% | 21 | | mid70% | distal50% | mid90% | | | yes | | | |
| Shanmugam | 50 | M | Present | Present | | Present | 168 | 78 | 27.6 | awmi+iwmi | class I | elevated | | lysed | ventricular | | 47% | 16 | | distal90% | distal99% | | | yes | | | | |
| Chandrasekar | 47 | M | | Present | Present | | 170 | 84 | 29 | awmi+iwmi | Class II | elevated | yes | lysed | ventricular | | 38% | 21 | | distal90% | | mid90% | | yes | | | | |
| Bagavathi | 55 | F | Present | | | Present | 161 | 73 | 28.1 | awmi+iwmi | class I | elevated | | lysed | | | 43% | 16 | | distal90% | ostial70% | mid90% | | | yes | | | |
| Rekha | 43 | F | Present | | | Present | 154 | 68 | 28.6 | awmi+iwmi | class I | elevated | | lysed | | | 45% | 16 | | proximal50% | | | | yes | | | | |
| Shankar | 51 | M | | Present | Present | | 171 | 79 | 27 | awmi+iwmi | class II | elevated | | lysed | ventricular | | 39% | 21 | | | | mid90% | | yes | | | | |
| Kumar | 45 | m | | | Present | Present | 166 | 77 | 28 | awmi+iwmi | class I | elevated | | lysed | | | 40% | 16 | | distal50% | | | | yes | | | | |
| Lakshmanan | 50 | m | | Present | Present | Present | 170 | 82 | 28.3 | awmi+iwmi | class I | elevated | | lysed | | | 41% | 16 | | prox100% | distal70% | | | yes | | | | |
| Vijayakumar | 52 | m | | Present | Present | | 167 | 81 | 29 | awmi+iwmi | class II | elevated | yes | lysed | ventricular | | 37% | 21 | | mid70% | distal70% | | | yes | | | | |
| Manoharan | 50 | m | Present | Present | | | 165 | 73 | 26.8 | awmi+iwmi | class I | elevated | | lysed | | | 42% | 16 | | mid70% | | | | yes | | | | |
| Raja | 40 | m | | Present | Present | Present | 169 | 81 | 28.4 | awmi+iwmi | class II | elevated | | lysed | | | 43% | 16 | | mid luminal irregularities | | | | yes | | | | |
| Gunasekaran | 56 | m | Present | | | | 164 | 76 | 28.3 | awmi+iwmi | class II | elevated | | lysed | | | 41% | 16 | | mid&distal70% | | proxi90% | | yes | | | | |
| Parthasarathy | 55 | m | Present | | | | 165 | 73 | 26.8 | awmi+iwmi | class I | elevated | | lysed | | | 40% | 16 | | | | proxi90% | | yes | | | | |
| Vijayakumar | 60 | m | | Present | | Present | 169 | 90 | 31.5 | awmi+iwmi | class I | elevated | | lysed | | | 40% | 16 | | mid100% | prox70% | prox70% | | | | yes | | |
| Munivel | 45 | m | | | Present | | 171 | 82 | 28 | awmi+iwmi | class II | elevated | | lysed | | | 41% | 16 | | prox99% | | prox50% | | yes | | | | |
| Rabhakanthan | 60 | m | Present | | | Present | 170 | 88 | 30.4 | awmi+iwmi | class I | elevated | | lysed | | | 40% | 21 | | mid90% | prox70% | mid90% | | yes | | | | |
| Periyasamy | 54 | m | Present | | Present | | 165 | 79 | 29 | awmi+iwmi | class II | elevated | | lysed | | | 45% | 16 | | mid70% | prox70% | distal100% | | | yes | | | |
| Susaimary | 59 | f | Present | | | | 152 | 68 | 29.4 | awmi+iwmi | class I | elevated | | lysed | | | 40% | 21 | | mid70% | distal90% | prox100% | | | | yes | | |
| Chandra | 50 | f | | Present | | Present | 154 | 69 | 29.1 | awmi+iwmi | class I | elevated | | lysed | | | 43% | 16 | | ostial50% | | | | yes | | | | |
| Ravikumar | 53 | m | Present | | Present | | 167 | 74 | 26.6 | awmi+iwmi | class II | elevated | yes | lysed | ventricular | | 34% | 21 | | distal 90% | | | | yes | | | | |
| Adrin | 43 | m | | Present | Present | | 168 | 76 | 26.9 | awmi+iwmi | class I | elevated | | lysed | | | 45% | 16 | | mid99% | | | | yes | | | | |
| Gopi | 55 | m | | Present | Present | | 173 | 81 | 27 | awmi+iwmi | Class III | elevated | yes | lysed | ventricular | | 30% | 25 | | prox50% | | prox70%&mid70% | | | yes | | | |
| Shahul hameed | 46 | m | Present | | | Present | 175 | 86 | 28.1 | awmi+iwmi | class II | elevated | yes | lysed | ventricular | | 34% | 21 | distal3 | mid50%% | | prox99% | | yes | | | | |
| Sivagnanam | 37 | m | | Present | Present | | 167 | 88 | 31.6 | awmi+iwmi | class I | elevated | | lysed | | | 40% | 16 | distal7 | mid70% | | mid90% | | yes | | | | |
| Chellappan | 57 | m | Present | | | Present | 170 | 70 | 24.2 | awmi+iwmi | class II | elevated | | lysed | ventricular | | 37% | 21 | | prox70% | prox70% | mid90% | | | yes | | | |
| Saroja | 67 | f | Present | | | | 155 | 67 | 27.9 | awmi+iwmi | Class III | elevated | yes | not lyse | ventricular | | 33% | 21 | | | | mid80% | | yes | | | | |
| Rajendran | 52 | m | | Present | Present | | 165 | 67 | 24.6 | awmi+iwmi | class I | elevated | | lysed | | | 41% | 16 | | mid90% | | | | yes | | | | |
| Jayakumar | 62 | m | Present | | | Present | 169 | 76 | 26.6 | awmi+iwmi | class I | elevated | | lysed | | yes | 42% | 16 | | prox90% | prox70% | | | yes | | | | |
| Karuppaiah | 52 | m | | Present | Present | | 171 | 78 | 26.7 | awmi+iwmi | class II | elevated | | lysed | | | 39% | 21 | | prox70% | prox70% | mid90% | | | yes | | | |
| Elango | 60 | m | | Present | Present | | 168 | 70 | 24.8 | awmi+iwmi | class I | elevated | | lysed | | | 41% | 16 | | distal 90% | | prox40% | | yes | | | | |
| Padmavathi | 65 | f | Present | | | Present | 161 | 67 | 25.8 | awmi+iwmi | class II | elevated | | lysed | | | 37% | 21 | | | | prox70% | | yes | | | done | |
| Stephen | 58 | m | Present | | Present | | 165 | 72 | 26.4 | awmi+iwmi | class I | elevated | | lysed | | | 41% | 16 | | distal 90% | | | | yes | | | | |
| Devaraj | 50 | m | | Present | Present | | 168 | 78 | 27.6 | awmi+iwmi | Class III | elevated | yes | lysed | ventricular | | 31% | 25 | | distal 90% | | prox 70% | | yes | | | | |
| Kannayiram | 68 | m | Present | | | | 169 | 74 | 25.9 | awmi+iwmi | class II | elevated | | lysed | | | 38% | 21 | | mid 70% | | | | yes | | | | |
| Singaraj | 59 | m | | Present | Present | | 166 | 69 | 25 | awmi+iwmi | class I | elevated | | lysed | | | 43% | 16 | | distal 90% | | | | yes | | | | |
| Eivendran | 52 | m | | Present | Present | Present | 167 | 84 | 30.2 | awmi+iwmi | class II | elevated | | lysed | | | 35% | 21 | | mid 70% | prox 50% | | | yes | | | | |
| Senthilvel | 53 | m | | Present | Present | Present | 169 | 87 | 30.5 | awmi+iwmi | class I | elevated | | lysed | | | 41% | 16 | | distal 90% | | | | yes | | | | |
| Jegannathan | 60 | m | Present | | Present | | 167 | 71 | 25.5 | awmi+iwmi | Class III | elevated | yes | lysed | ventricular | | 32% | 21 | | mid 50% & distal 70% | | prox 70% | | | yes | | | |
| Jayaraman | 53 | m | | Present | Present | | 163 | 69 | 26 | awmi+iwmi | class I | elevated | | lysed | | | 42% | 16 | | distal 90% | | | | yes | | | | |
| Gopinathan | 54 | m | Present | | Present | | 166 | 68 | 24.7 | awmi+iwmi | class II | elevated | | lysed | | | 45% | 16 | | distal 70% | | | | yes | | | | |
| Ramalakshmi | 63 | f | Present | | | Present | 162 | 83 | 31.6 | awmi+iwmi | class I | elevated | | lysed | | | 40% | 16 | | distal 90% | | | | yes | | | | |
| Prakasam | 60 | f | | Present | | | 159 | 73 | 28.9 | awmi+iwmi | class II | elevated | | lysed | ventricular | | 36% | 21 | | mid 90% | | | | yes | | | | |
| Munvar khan | 46 | m | Present | | Present | Present | 164 | 86 | 32 | awmi+iwmi | Class III | elevated | yes | lysed | | yes | 30% | 25 | | mid 70% | | prox 70% | | yes | | | done | |
| Indhumathi | 65 | f | Present | Present | | | 157 | 74 | 30 | awmi+iwmi | class I | elevated | | lysed | | | 43% | 16 | | distal 90% | | | | yes | | | | |
| Kannayiram | 65 | m | | Present | Present | | 169 | 81 | 28.4 | awmi+iwmi | class II | elevated | yes | not lyse | ventricular | | 37% | 21 | | mid 90% | | | | yes | | | | |
| Vadivambal | 64 | f | Present | Present | | | 170 | 76 | 26.2 | awmi+iwmi | class I | elevated | | lysed | | | 41% | 16 | | distal 90% | | | | yes | | | | |
| Chelladurai | 57 | m | | Present | Present | Present | 168 | 81 | 28.7 | awmi+iwmi | class II | elevated | | lysed | | | 45% | 16 | | distal 70% | | | | yes | | | | |
| Arockia doss | 52 | m | | Present | Present | | 167 | 79 | 28.4 | awmi+iwmi | class I | elevated | | not lysed | | | 41% | 16 | | prox70%&mid100% | | | | yes | | | pri PCI | |
| Patchaimuthu | 70 | m | Present | | Present | | 169 | 83 | 29.1 | awmi+iwmi | class IV | elevated | yes | lysed | ventricular | | 30% | 25 | | distal 90% | | | | yes | | | | yes |
| Sundar | 43 | m | | Present | Present | | 165 | 72 | 26.4 | awmi+iwmi | class IV | elevated | yes | lysed | ventricular | | 31% | 25 | | distal 90% | | | | yes | | | | yes |

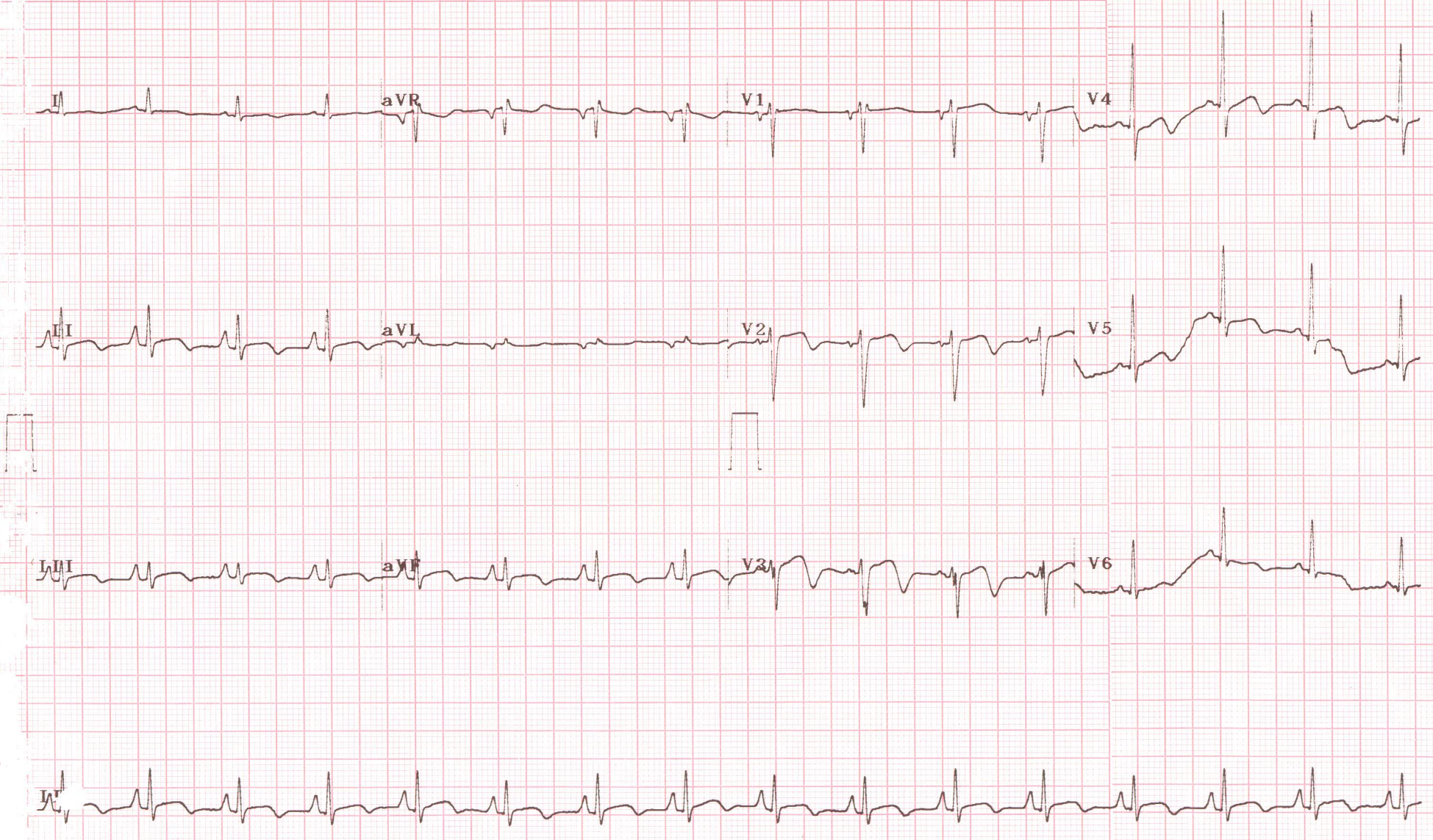
2012-11-22 08:49:55 3Channel+1 Rhythm Report Hospital:stanley
Confirmed by:g

ID:
Name:
Age:Years
Sex:
H:cm/W:0kg

Heart Rate : 93 bpm
PR Int.: 114 ms
QRS Dur.: 82 ms
QT/QTc: 348/437 ms
P-R-T axes: 80 24 70

** Analysis Result ** (To be finally confirmed by cardiologist)
Normal Sinus Rhythm
Normal Axis
RAE(Right Atrial Enlargement)
[ Moderately Abnormal ECG ]

Ramalakshmi
63/F
22/11/12




0.1Hz- 40Hz,AC50Hz.PM All Channels:10.0mm/mV.25.0mm/sec. CardioTouch6.08C.30 Bionet Co.,Ltd.

2012-11-21 08:48:31 3Channel+1 Rhythm Report Hospital:stanley

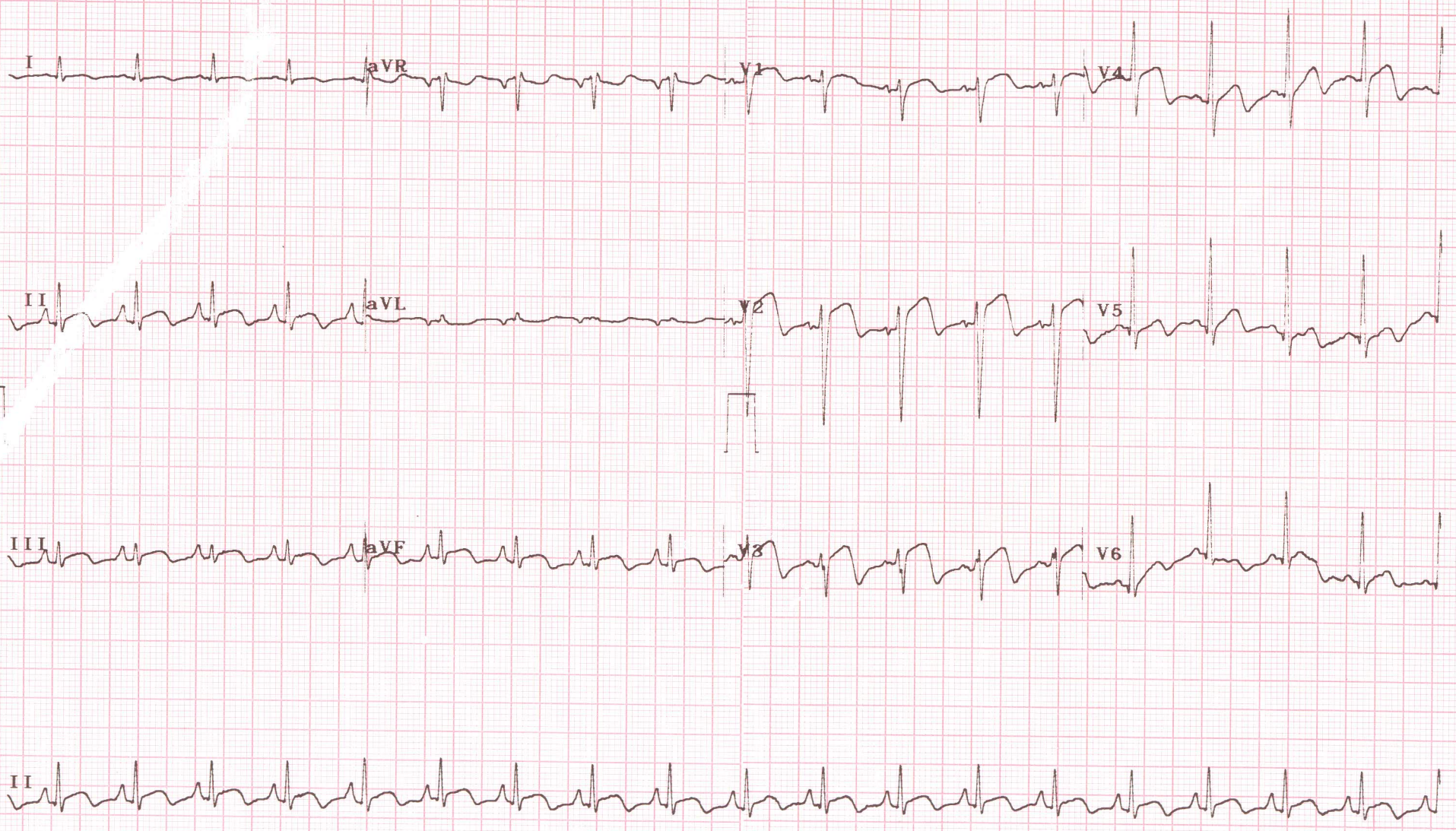
Confirmed by:g

ID:
Name:
Age:Years
Sex:
H:cm/W:0kg

Heart Rate : 112 bpm
PR Int.: 110 ms
QRS Dur.: 76 ms
QT/QTc: 304/418 ms
P-R-T axes: 82 31 75

** Analysis Result ** (To be finally confirmed by cardiologist)
Sinus Tachycardia(Heart Rate:100-130)
Normal Axis
[ Minimally Abnormal or Normal Variation ECG ]

Ramadhani 63/F
2/11/12
8.40am




0.1Hz- 40Hz,AC50Hz.PM All Channels:10.0mm/mV.25.0mm/sec. CardioTouch6.08C.30 Bionet Co.,Ltd.

2012-11-16 11:42:32 3Channel+1 Rhythm Report Hospital:

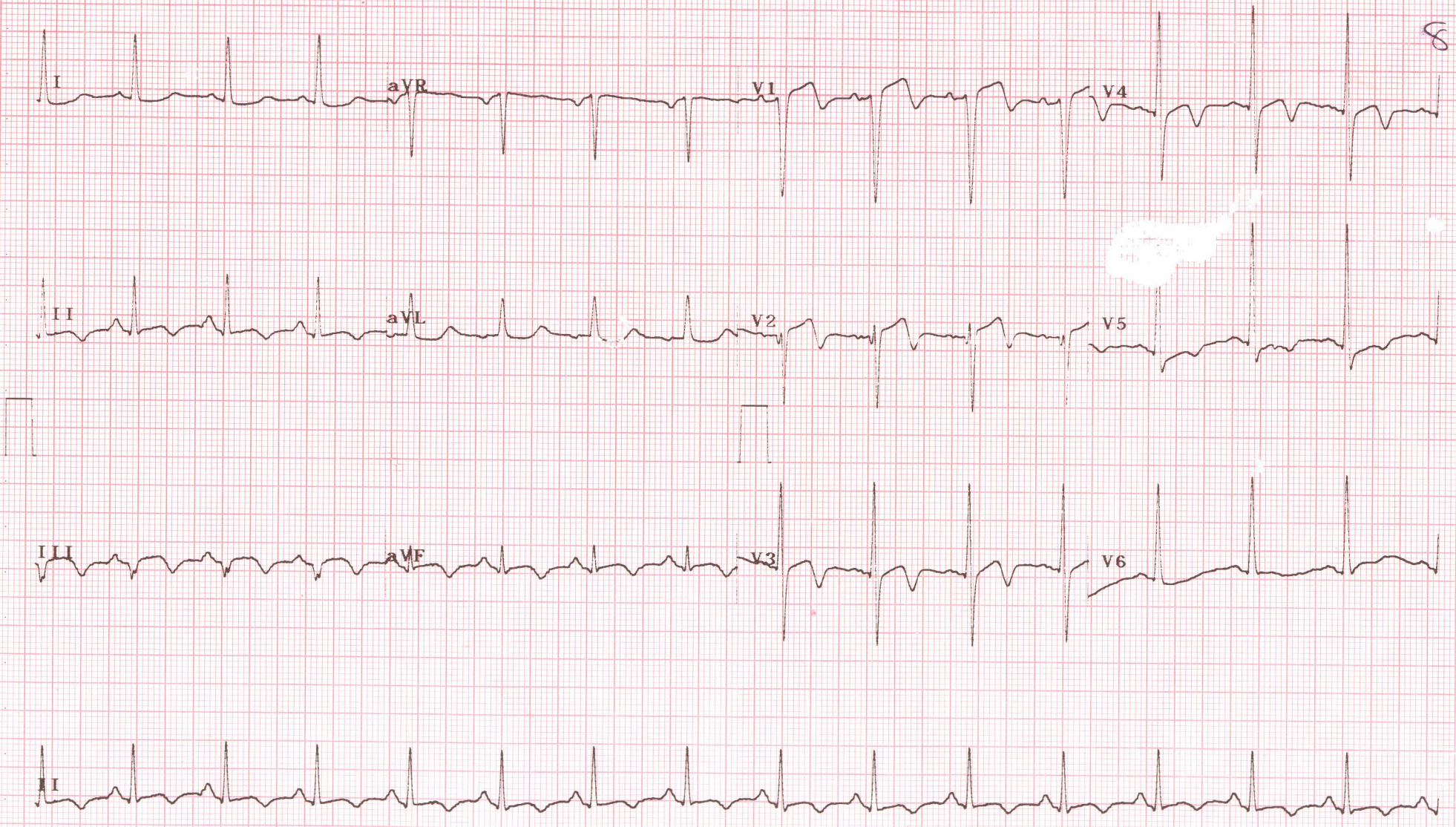
Confirmed by:

ID:
Name:
Age:Years
Sex:
H:cm/W:0kg

Heart Rate : 90 bpm
PR Int.: 132 ms
QRS Dur.: 100 ms
QT/QTc: 378/461 ms
P-R-T axes: 66 14 -70

** Analysis Result ** (To be finally confirmed by cardiologist)
Normal Sinus Rhythm
Normal Axis
LVH(Left Ventricular Hypertrophy)
T wave inversion, possible Myocardial ischemia(Anteroseptal)
[ Moderately Abnormal ECG ]

Chandrasekaran
16/11/12
8.30am




0.1Hz- 40Hz,AC50Hz.PM All Channels:10.0mm/mV.25.0mm/sec. CardioTouch6.08C.30 Bionet Co.,Ltd.

15-Nov-12 8:41:29 AM

govt stanley hospital

cardiology (35)

Chandraiya 50/m

15/11/12 at 8-40am

| | | |
|---|---|---|
| ate | 82 | . Age not entered, assumed to be 50 years old for purpose of ECG interpretation |
| | | . Atrial flutter with predominant 4:1 AV block.....A-rate 333, multiple Ps |
| PR | | . Probable anteroseptal infarct, recent.....Q, ST>0.15mV, T neg, V1-V2 |
| RSD | 104 | . Minimal ST elevation, inferior leads.....ST >0.06mV, II III aVF |
| QT | 436 | . Prolonged QT interval.....QTc >500mS |
| QTc | 510 | |

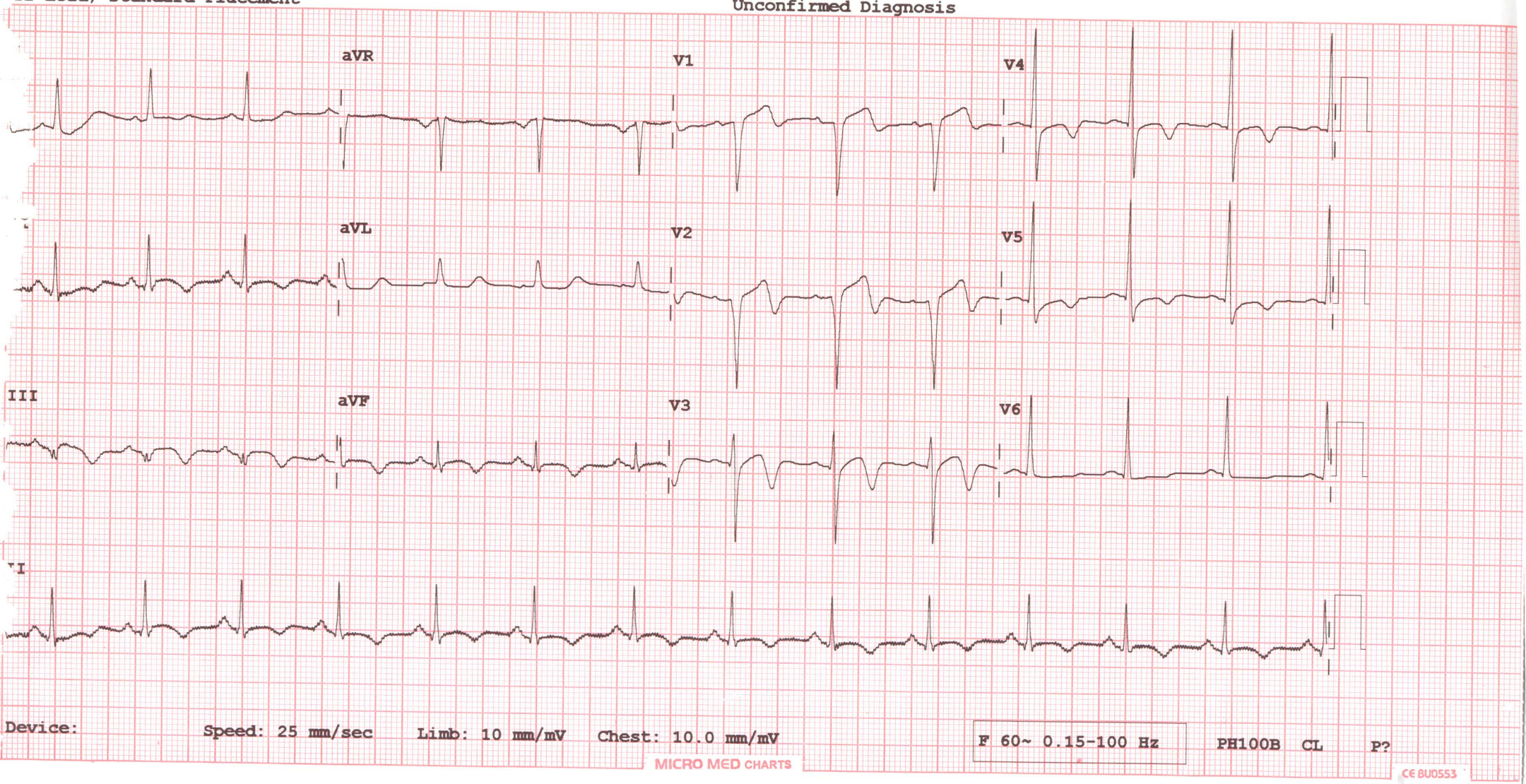
-AXIS--

| | |
|---|---|
| QRS | 3 |
| | -73 |

- ABNORMAL ECG -

12 Lead; Standard Placement

Unconfirmed Diagnosis




Device: Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10.0 mm/mV

F 60~ 0.15-100 Hz PH100B CL P?

MICRO MED CHARTS

CE BU0553

11-Nov-12 8:06:21 AM

govt stanley hospital

cardiology (35)

Kadarkarai 8/11/12
8.05 am.
11/11/12

| | | |
|---|---|---|
| Rate | 121 | . Age not entered, assumed to be 50 years old for purpose of ECG interpretation |
| | | . Sinus tachycardia.....rate> 99 |
| PR | 150 | . Multiple ventricular premature complexes.....V complexes w/ short R-R intervls |
| QRSD | 87 | . Anterolateral infarct, acute (LAD).....ST >0.20mV, V2-V6,I,aVL |
| QT | 274 | . Baseline wander in lead(s) V2,V3,V4 |
| QTc | 389 | |

--AXIS--

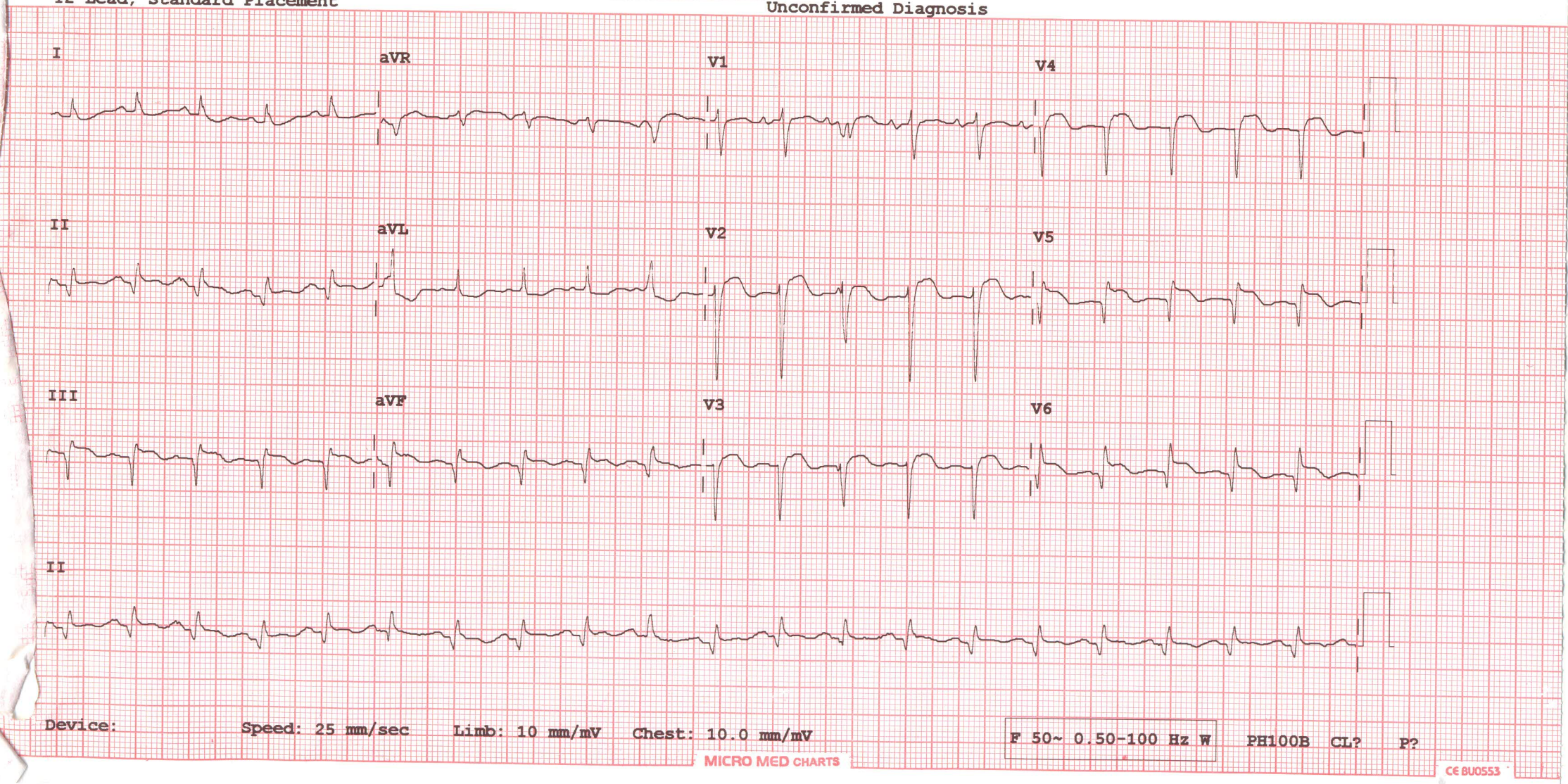
| | |
|---|---|
| P | 47 |
| QRS | 2 |
| T | 176 |

12 Lead; Standard Placement

- ABNORMAL ECG -

>>> Acute MI <<<

Unconfirmed Diagnosis




Device: Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10.0 mm/mV F 50~ 0.50-100 Hz W PH100B CL? P?

MICRO MED CHARTS

CE 8U0553

10-Nov-12 6:39:08 PM

govt stanley hospital

cardiology (35)

| | | |
|---|---|---|
| Rate | 130 | . Age not entered, assumed to be 50 years old for purpose of ECG interpretation |
| | | . Sinus tachycardia.....rate> 99 |
| PR | 140 | . Probable inferior infarct, recent.....Q>25mS, ST>0.07mV, T neg, II-aVF |
| QRSD | 107 | . Anterolateral infarct, acute (LAD).....ST >0.20mV, V2-V6,I,aVL |
| QT | 337 | |
| QTc | 496 | |

Thanammal - 85/F

--AXIS--

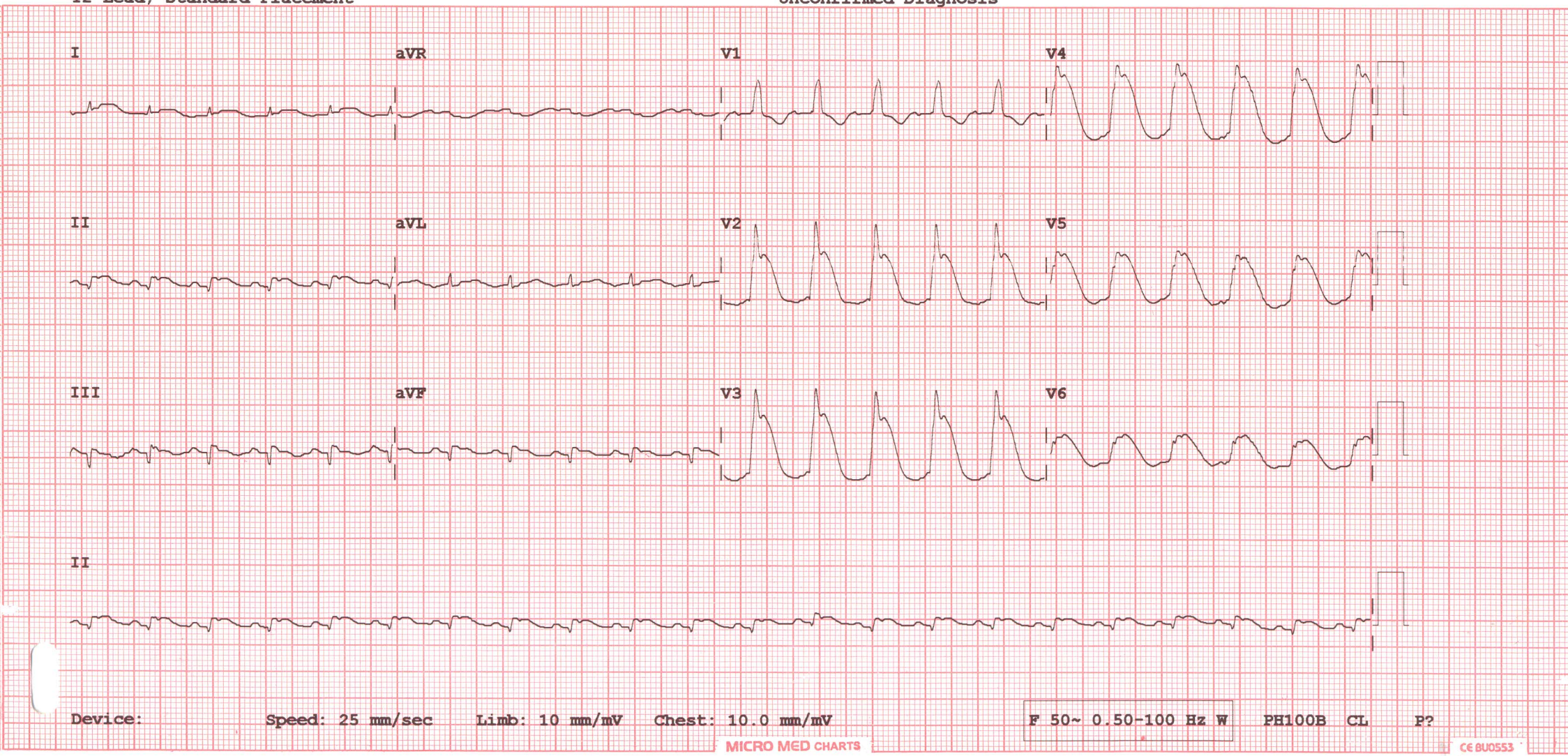
| | |
|---|---|
| P | 90 |
| QRS | 47 |
| T | 4 |

- ABNORMAL ECG -

>>> Acute MI <<<

12 Lead; Standard Placement

Unconfirmed Diagnosis




Device: Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10.0 mm/mV F 50~ 0.50-100 Hz W PH100B CL P?

MICRO MED CHARTS

CE BU0553

07-Nov-12 7:55:55 AM

govt stanley hospital

cardiology (35)

③

| | | |
|---|---|---|
| Rate | 87 | . Age not entered, assumed to be 50 years old for purpose of ECG interpretation |
| | | . Sinus rhythm.....normal P axis, V-rate 50- 99 |
| PR | 206 | . Borderline prolonged PR interval.....PR >202, V-rate 50- 90 |
| QRSD | 80 | . Inferior infarct, old.....Q >35mS, II III aVF |
| QT | 348 | . Anterior infarct, acute (LAD).....ST >0.25mV, V2-V5 |
| QTc | 419 | . Baseline wander in lead(s) V1 |

--AXIS--

| | |
|---|---|
| P | 43 |
| QRS | -56 |
| T | 87 |

- ABNORMAL ECG -

>>> Acute MI <<<

12 Lead; Standard Placement

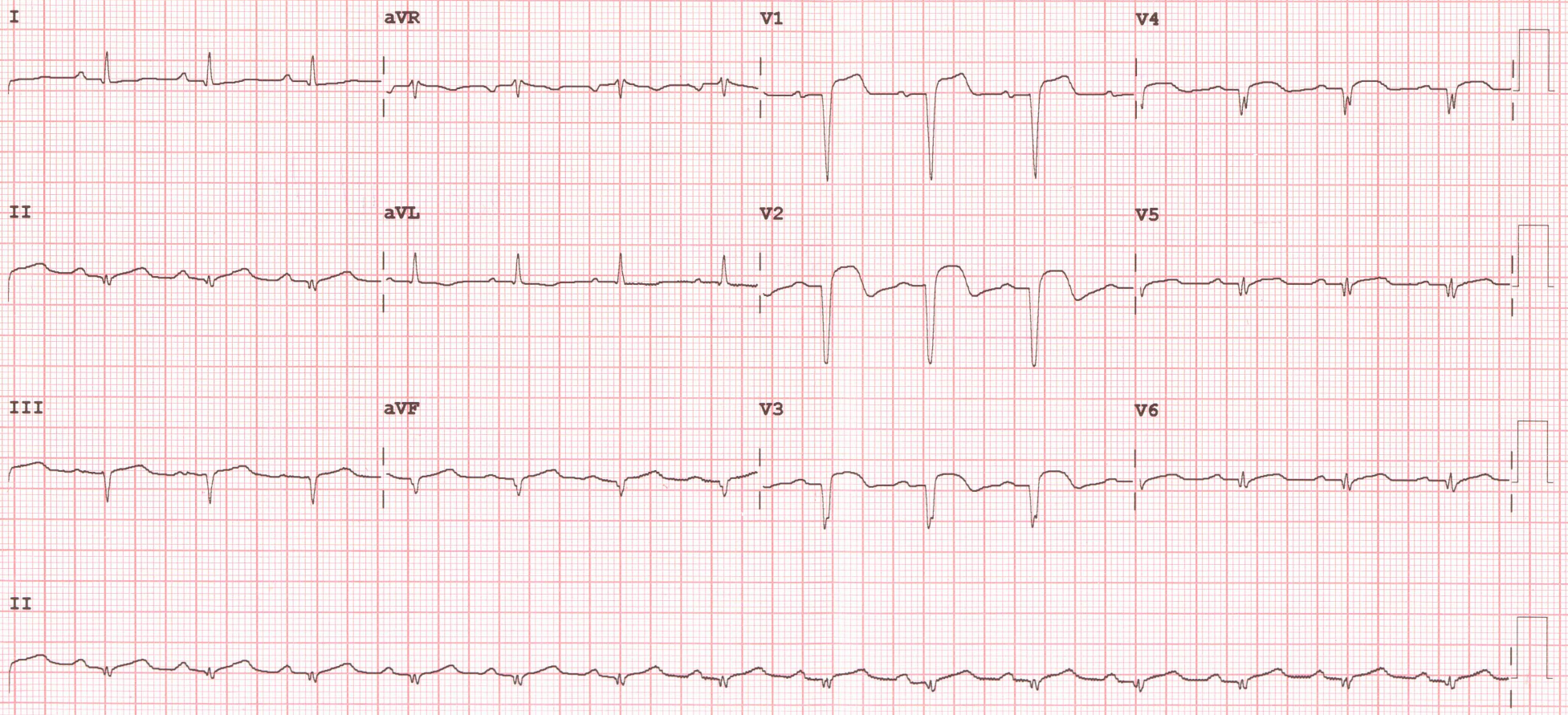
Unconfirmed Diagnosis

Gopalakrishnan

59/M

Time: 7.55 AM

7-11-12




Device: Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10.0 mm/mV F 60~ 0.15-100 Hz PH100B CL P?

MICRO MED CHARTS

CE BU0553

06-Sep-12 12:02:15 PM

govt stanley hospital

cardiology (35)

Post SIC

12.00 PM

Jayaraman

53/M

6/9/12

| | | |
|---|---|---|
| Rate | 98 | . Age not entered, assumed to be 50 years old for purpose of ECG interpretation |
| | | . Sinus rhythm.....normal P axis, V-rate 50- 99 |
| PR | 134 | . Inferior infarct, old.....Q >35mS, II III aVF |
| QRSD | 90 | . Extensive anterior infarct, acute (LAD).....ST >0.20mV, V1-V6 |
| QT | 337 | |
| QTc | 431 | |

--AXIS--

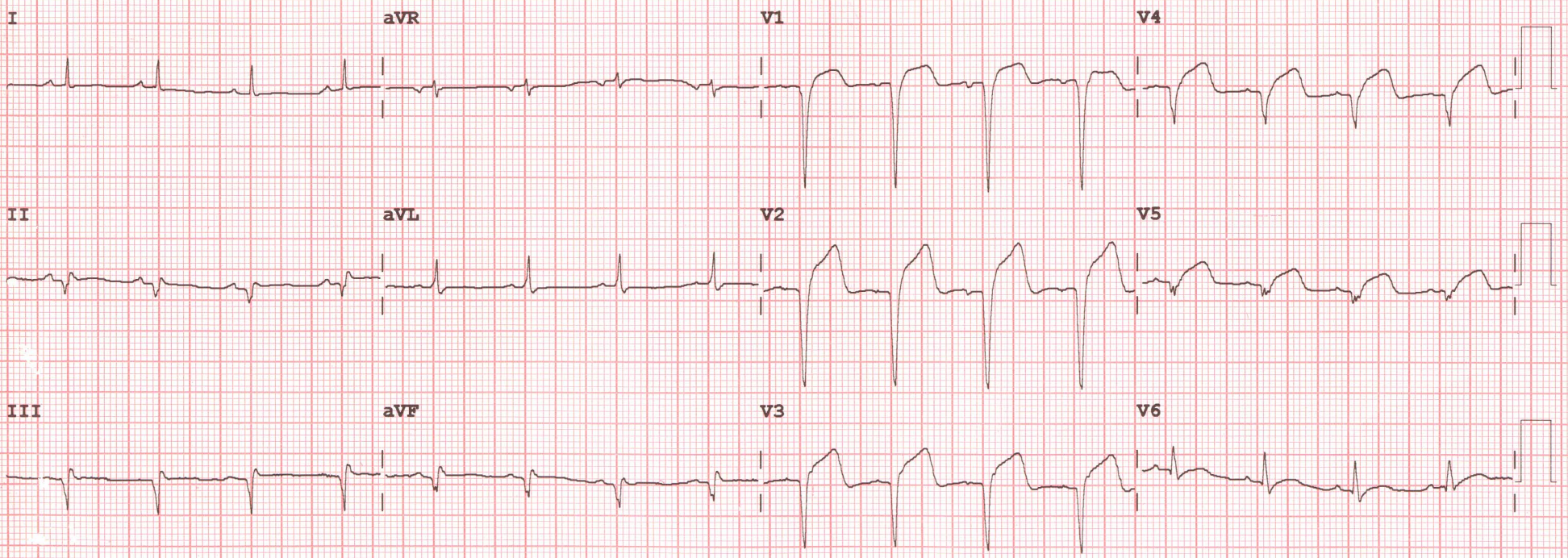
| | |
|---|---|
| P | 40 |
| QRS | -33 |
| T | 73 |

- ABNORMAL ECG -

>>> Acute MI <<<

12 Lead; Standard Placement

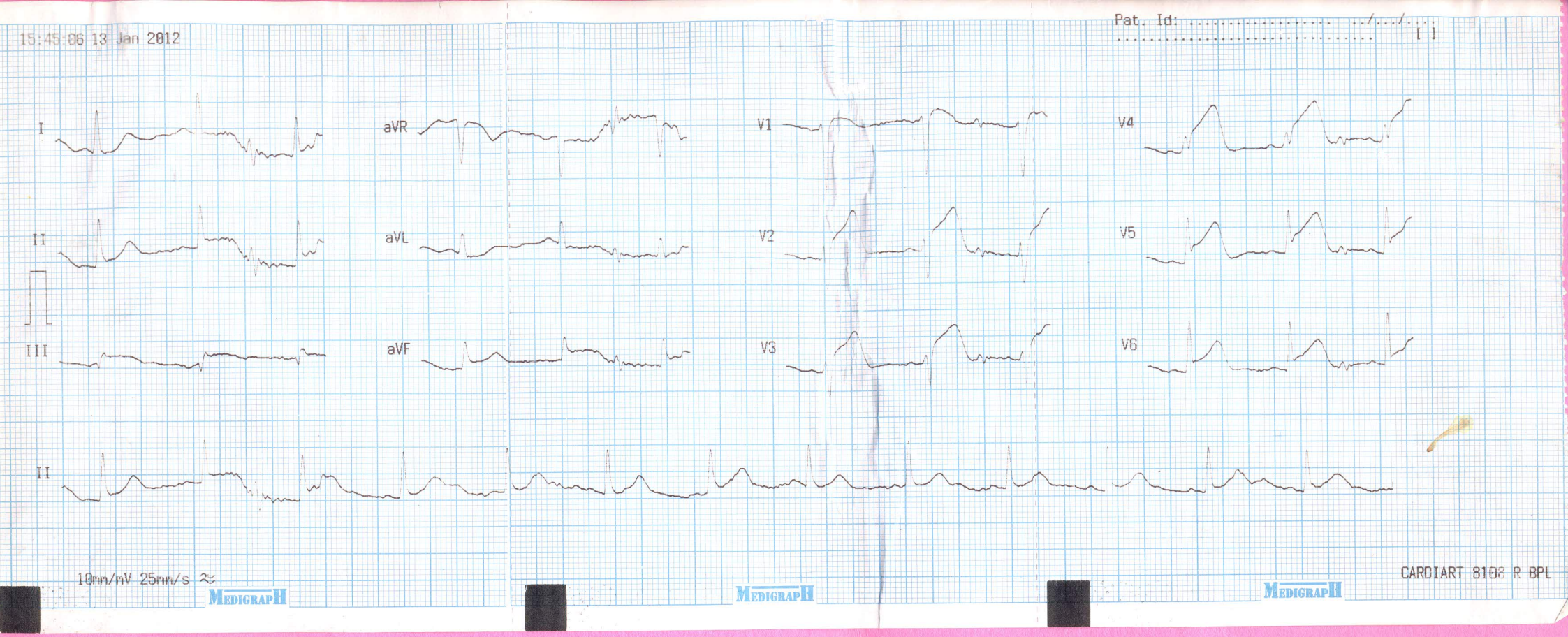
Unconfirmed Diagnosis




Device: Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10.0 mm/mV F 60~ 0.15-100 Hz PH100B CL P?

MICRO MED CHARTS

CE BU0553




G. Elumalai. 52/M.

13/7/12.

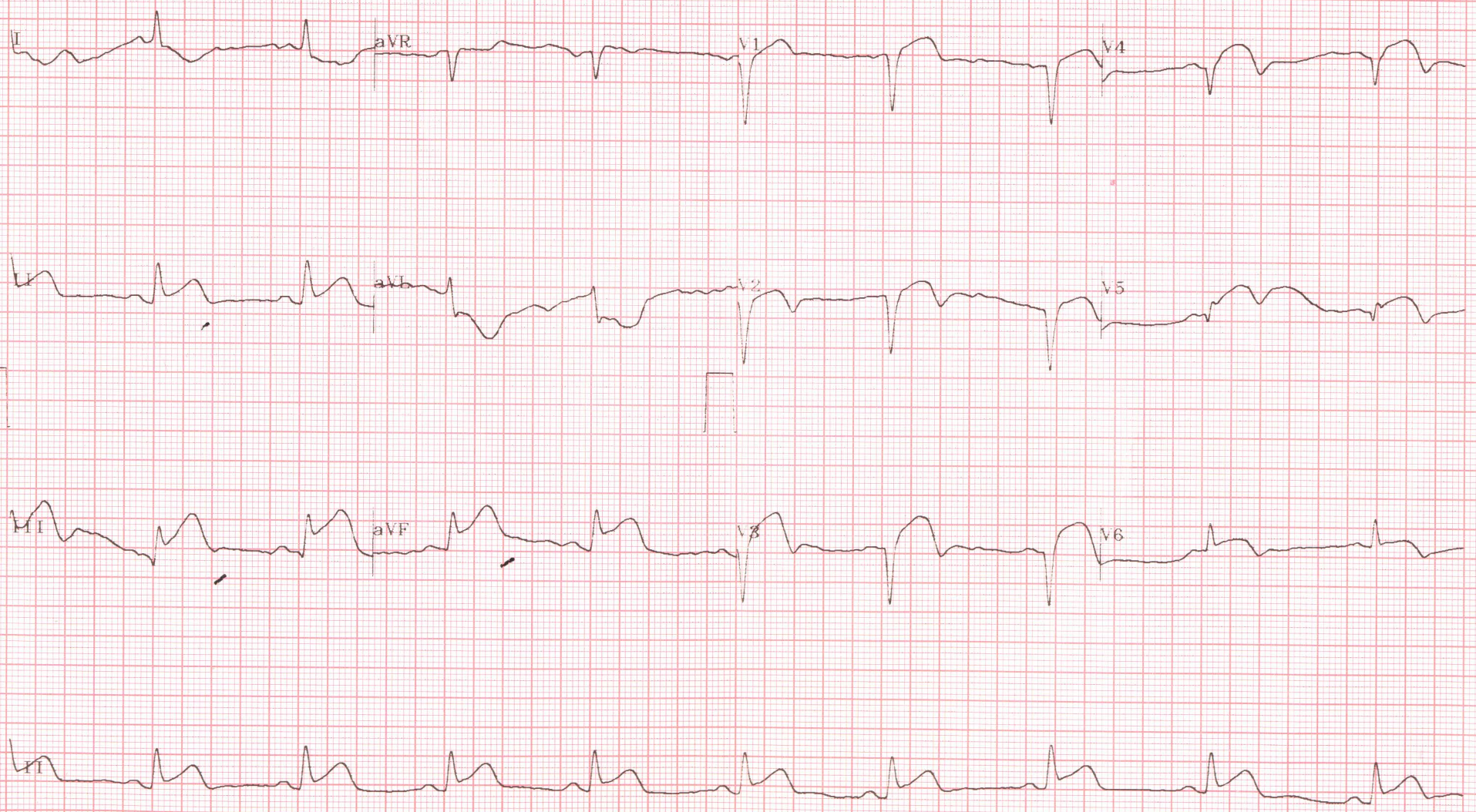
Prescribed by

ID :
Name:KANNAYARIAM
Age :65 yrs. / Sex :Male
Hei.: cm / Wei.: kg

Heart Rate: 57bpm
PR Int.: 142ms
QRS Dur.: 114ms
QT/QTc: 416/408ms
P-R-T axes 47 59 109

** Analysis Result ** (To be finally confirmed by cardiologist)
Sinus Bradycardia(HR:50-59)
Normal Axis
Ventricular preexcitation(WPW)
Anteroseptal MI
[ Markedly Abnormal ECG ]

Kannayiram 65/m
28/8/12



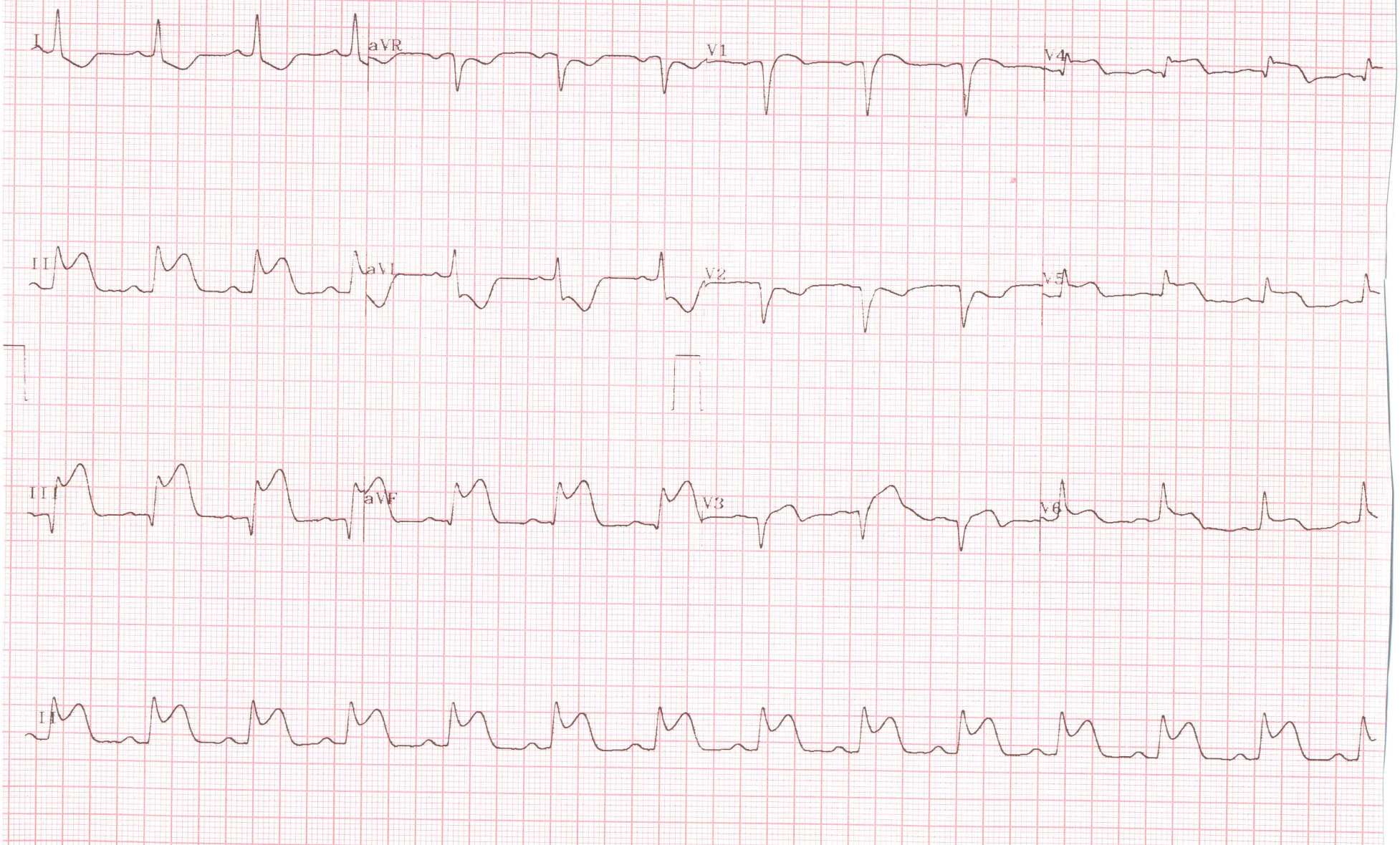

0.1Hz - 40Hz, AC 60Hz, EMG. All Channels:10mm/mV. 25mm/s. EKG2000 ver5.00.30 Bionet Co., Ltd

ID :
Name:
Age : yrs. / Sex :
Hei.: cm / Wei.: kg

Heart Rate: 81bpm
PR Int.: 168ms
QRS Dur.: 108ms
QT/QTc: 364/421ms
P-R-T axes 40 47 106

Prescribed by

** Analysis Result ** (To be finally confirmed by cardiologis
Normal Sinus Rhythm
Normal Axis
Anteroseptal MI
ST abnormality, possible subendocardial ischemia(High Lateral
[ Markedly Abnormal ECG ]




0.1Hz - 40Hz, AC 60Hz, EMG. All Channels:10mm/mV. 25mm/s. EKG2000 ver5.00.30 Bionet Co., Ltd

13-Sep-12 8:24:52 AM

govt stanley hospital

cardiology (35)

Kannayaram
68/M
13/9/12
8-30 A.M

| | | |
|---|---|---|
| Rate | 81 | . Age not entered, assumed to be 50 years old for purpose of ECG interpretation |
| | | . Sinus rhythm.....normal P axis, V-rate 50- 99 |
| PR | 150 | . Ventricular premature complex.....V complex w/ short R-R interval |
| QRSD | 79 | . Inferior infarct, recent.....Q>35mS, ST>0.07mV, T neg, II-aVF |
| QT | 433 | . Probable anterior infarct, age indeterminate.....Q >35mS, T neg, V2-V5 |
| QTc | 503 | . Prolonged QT interval.....QTc >500mS |

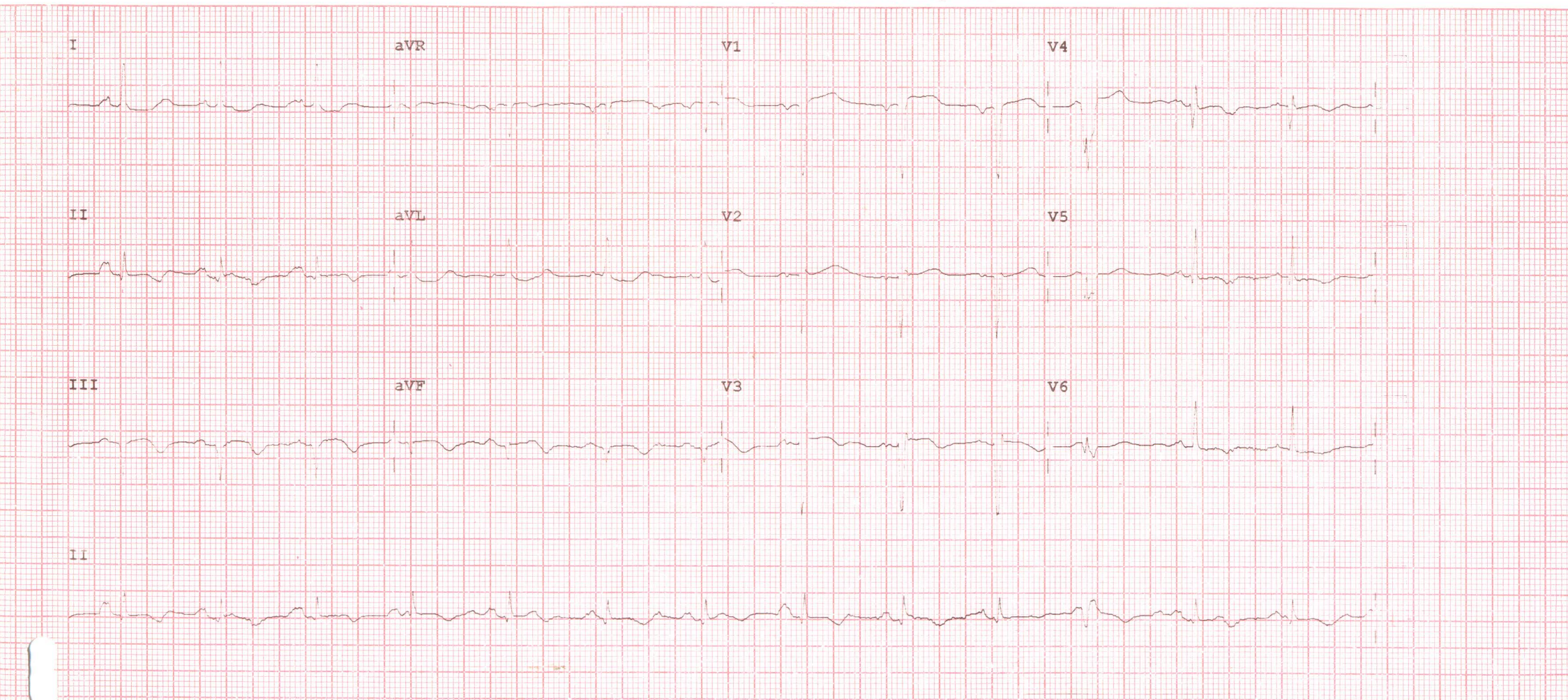
--AXIS--

| | |
|---|---|
| P | 51 |
| QRS | -19 |
| T | -73 |

- ABNORMAL ECG -

12 Lead; Standard Placement

Unconfirmed Diagnosis




Device: Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10.0 mm/mV F 50~ 0.50-100 Hz W PH100B CL? P?

6Chanr 2012-09-13 20:17:21 6Channel+1 Rhythm Report

Hospital:
Confirmed by:

*:ID:
N:Name:
L:Age:Years
N:Sex:
*:H:cm/W:0kg
[

Heart Rate : 60 bpm
PR Int.: 138 ms
QRS Dur.: 112 ms
QT/QTc: 426/427 ms
P-R-T axes: 56 47 37

** Analysis Result ** (To be finally confirmed by cardiologist)
Normal Sinus Rhythm
Low Voltage (Limb Leads)
Normal Axis
Ventricular preexcitation(WPW)
*** Axis may be incorrect due to low voltage.
[ Moderately Abnormal ECG ]

Sivagnanam
37/m
13/9/12

I
II
III
aVR
aVL
aVF
II

V1
V2
V3
V4
V5
V6

nnels:10. 0.1Hz- 40Hz,AC50Hz,EMG.PM All Channels:10.0mm/mV.25.0mm/sec.

CardioTouch6.08C.30 Bionet Co.,Ltd.

2012-09-05 14:57:46 6Channel+1 Rhythm Report Hospital:

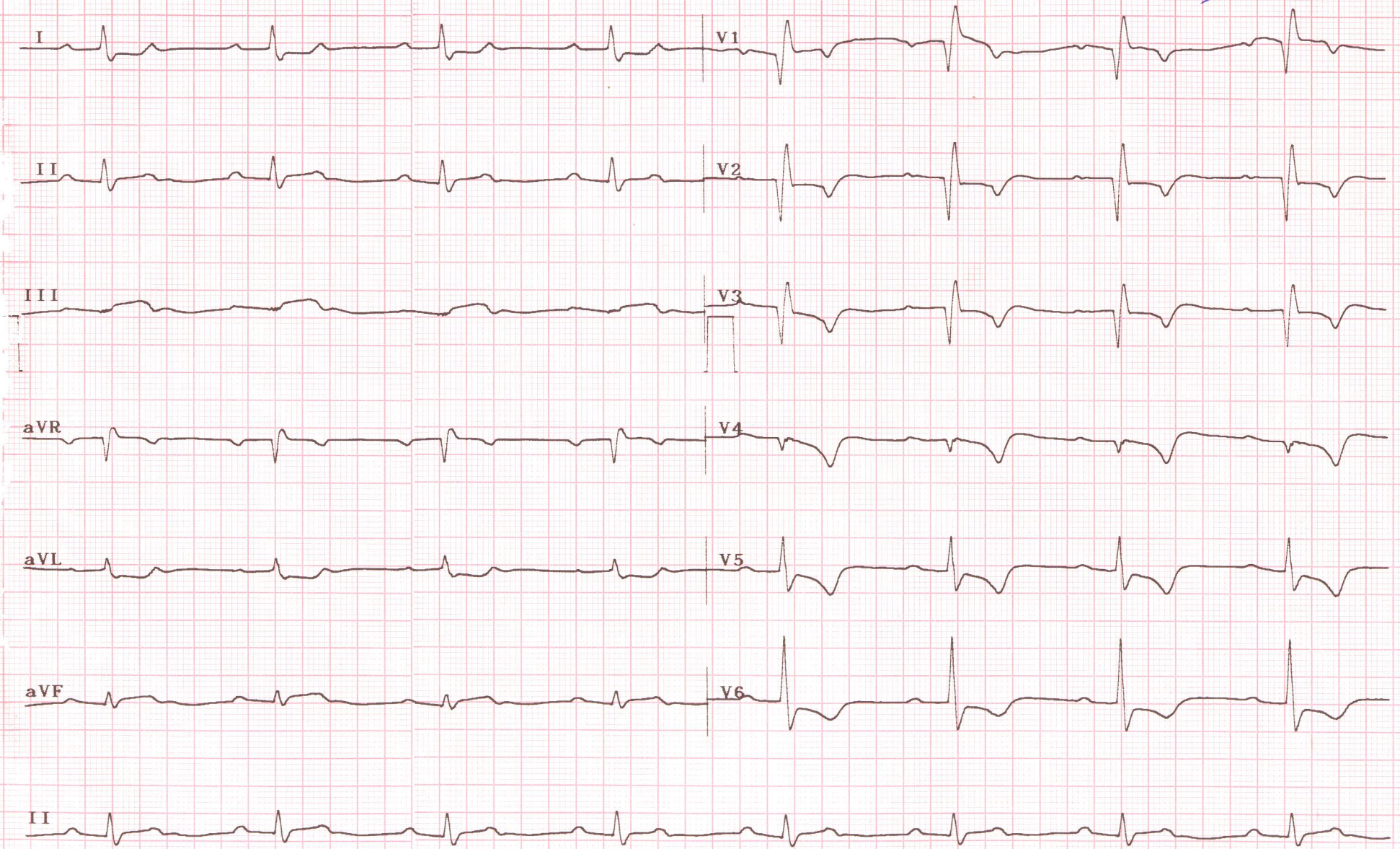
Confirmed by: (Post SR)

ID:
Name:
Age:Years
Sex:
H:cm/W:0kg

Heart Rate : 48 bpm
PR Int.: 290 ms
QRS Dur.: 128 ms
QT/QTc: 498/447 ms
P-R-T axes: 44 31 52

** Analysis Result ** (To be finally confirmed by cardiologist)
Sinus Bradycardia(Heart Rate<50)
AV Block I
Normal Axis
Nonspecific intraventricular coduction delay
Anteroseptal MI
[ Markedly Abnormal ECG ]

5/9/12
Decreased ICU/M
11.40AM 10.40 AM




0.1Hz- 40Hz,AC50Hz,EMG.PM All Channels:10.0mm/mV.25.0mm/sec. CardioTouch6.08C.30 Bionet Co.,Ltd.

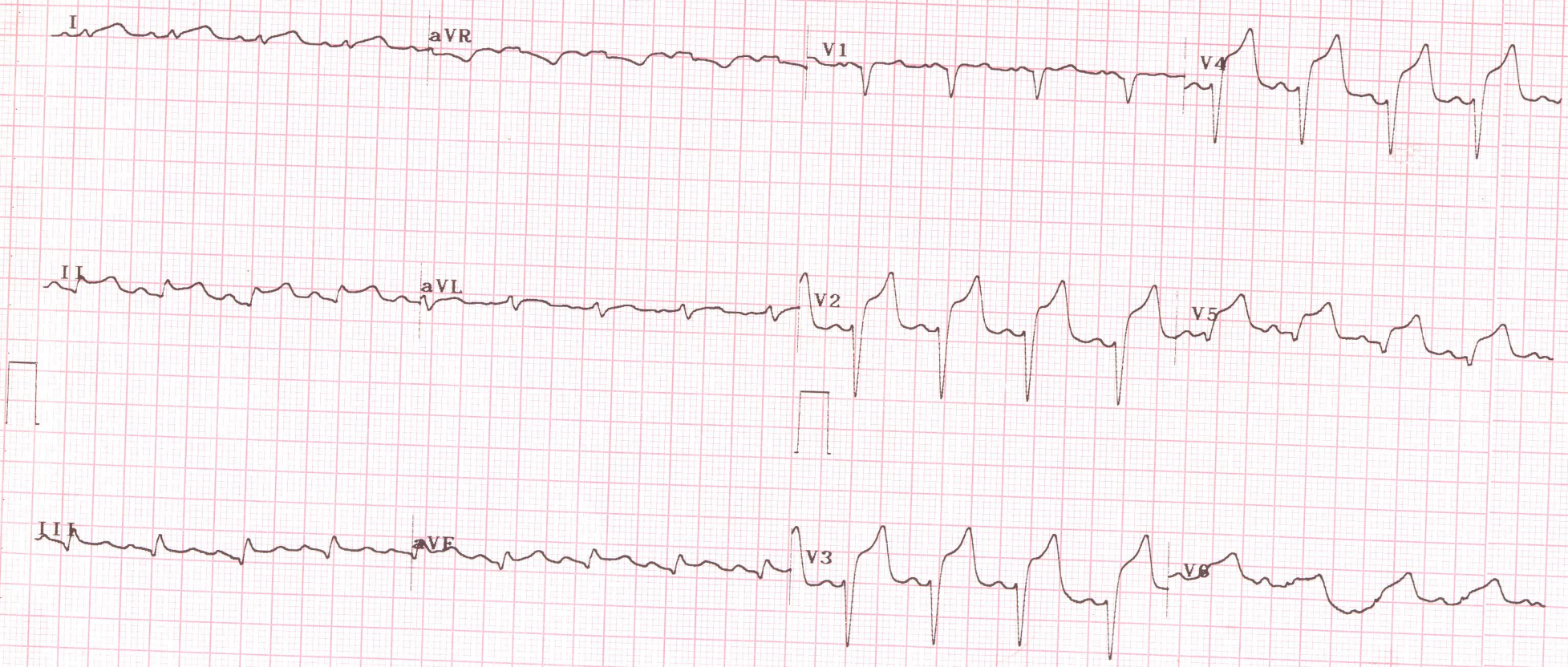
12-09-17 15:44:23 3Channel+1 Rhythm Report Hospital:stanley

ID:
Name:
Age:Years
Sex:
H:cm/W:0kg

Heart Rate : 110 bpm
PR Int.: * ms
QRS Dur.: 78 ms
QT/QTc: 346/450 ms
P-R-T axes:-90 64 2

Confirmed by:g

Dachamuthu
3.45pm
17/9/12




0.1Hz- 40Hz,AC50Hz,EMG.PM All Channels:10.0mm/mV,25.0mm/sec. CardioTouch6.08C.30 Bionet Co.,Ltd

17-Sep-12 4:39:29 PM

govt stanley hospital

cardiology (35)

| | | |
|---|---|---|
| Rate | 102 | . Age not entered, assumed to be 50 years old for purpose of ECG interpretation |
| | | . Sinus tachycardia.....rate> 99 |
| PR | 157 | . Inferior infarct, old.....Q >35mS, II III aVF |
| QRSD | 92 | . Anterior infarct, age indeterminate.....Q >35mS, T neg, in V2-V5 |
| QT | 347 | . Lateral leads are also involved.....lat Q or ST-T abnormalities |
| QTc | 453 | |

--AXIS--

| | |
|---|---|
| P | 76 |
| QRS | 85 |
| T | 34 |

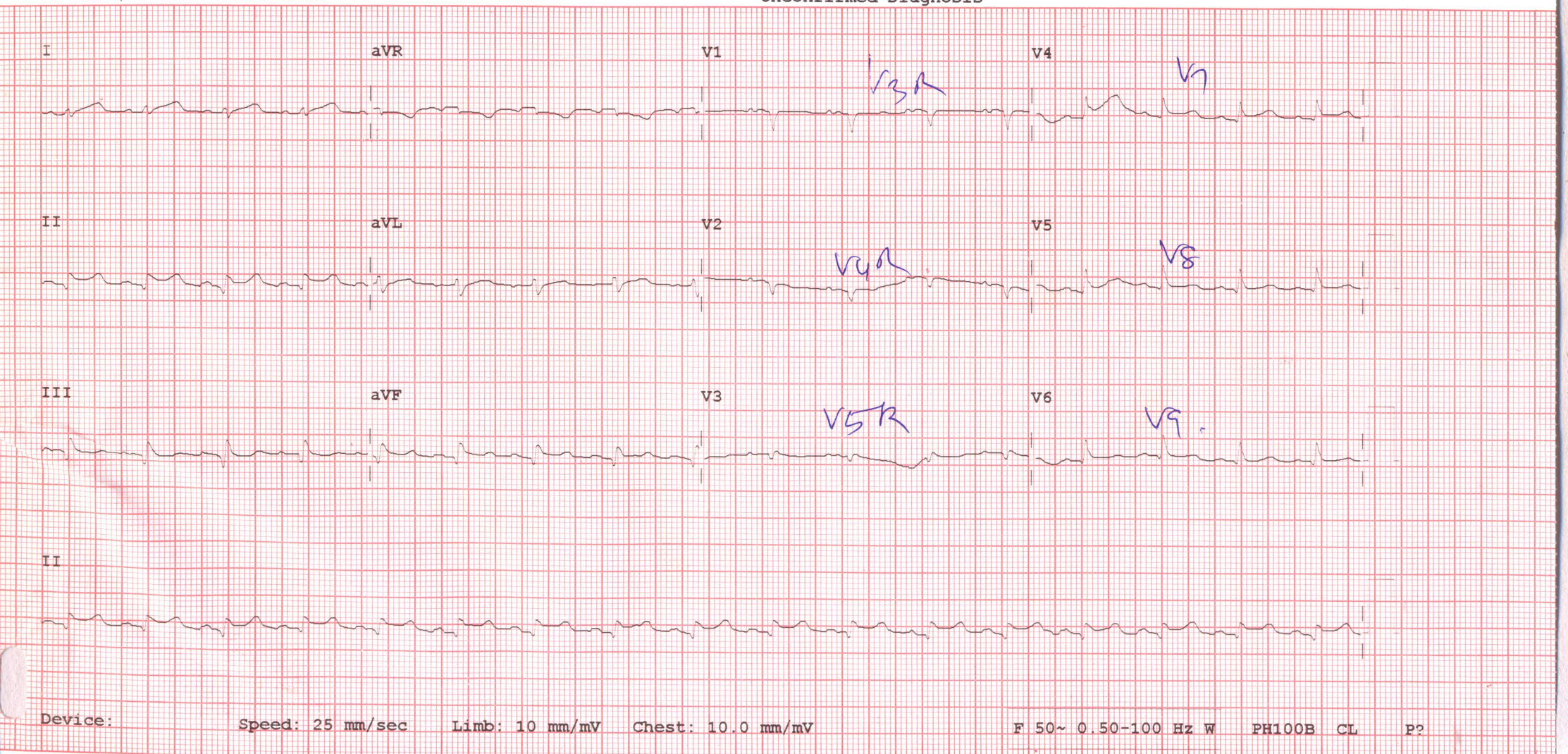
Patcheimuthu.

17/9/12 at 4.30pm

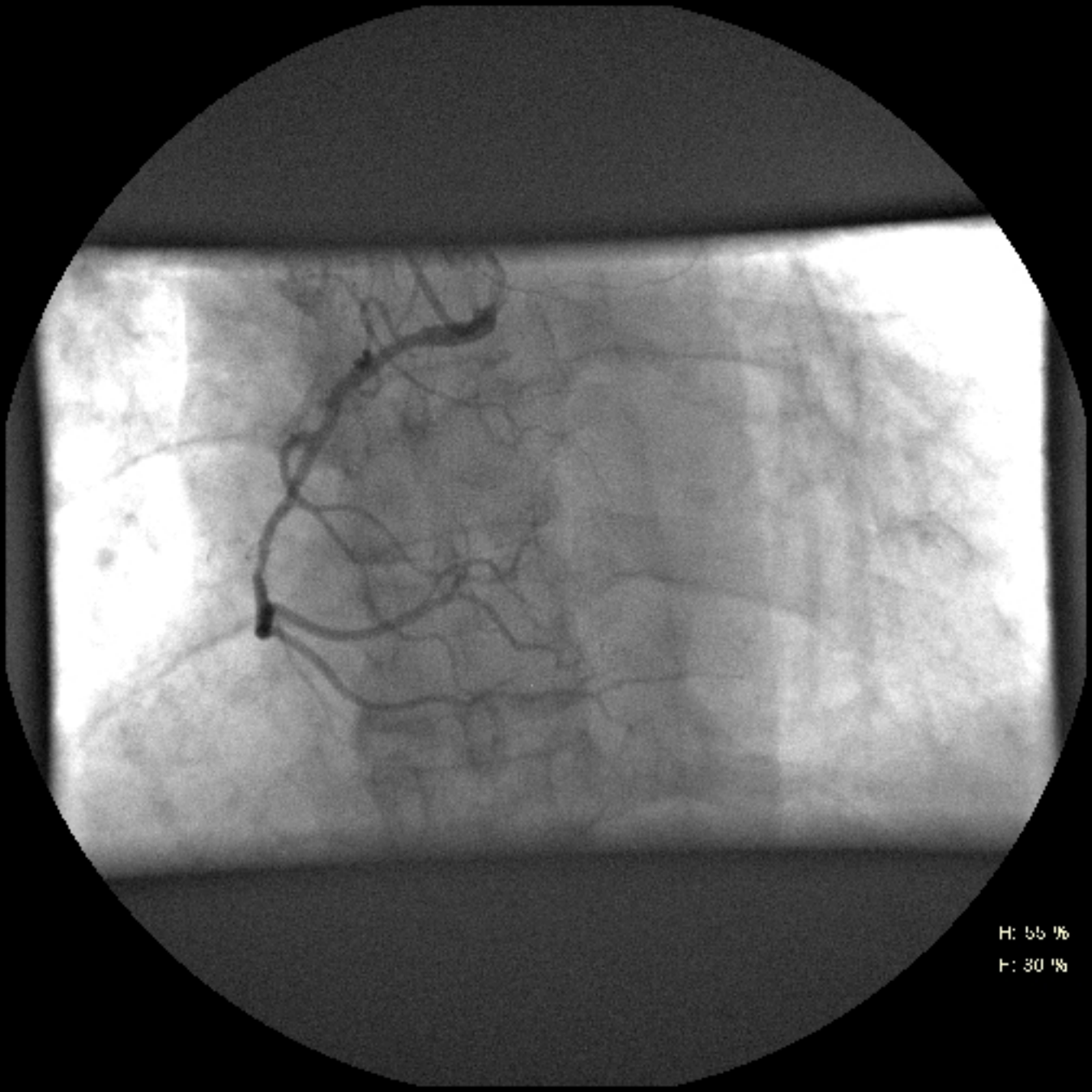
- ABNORMAL ECG -

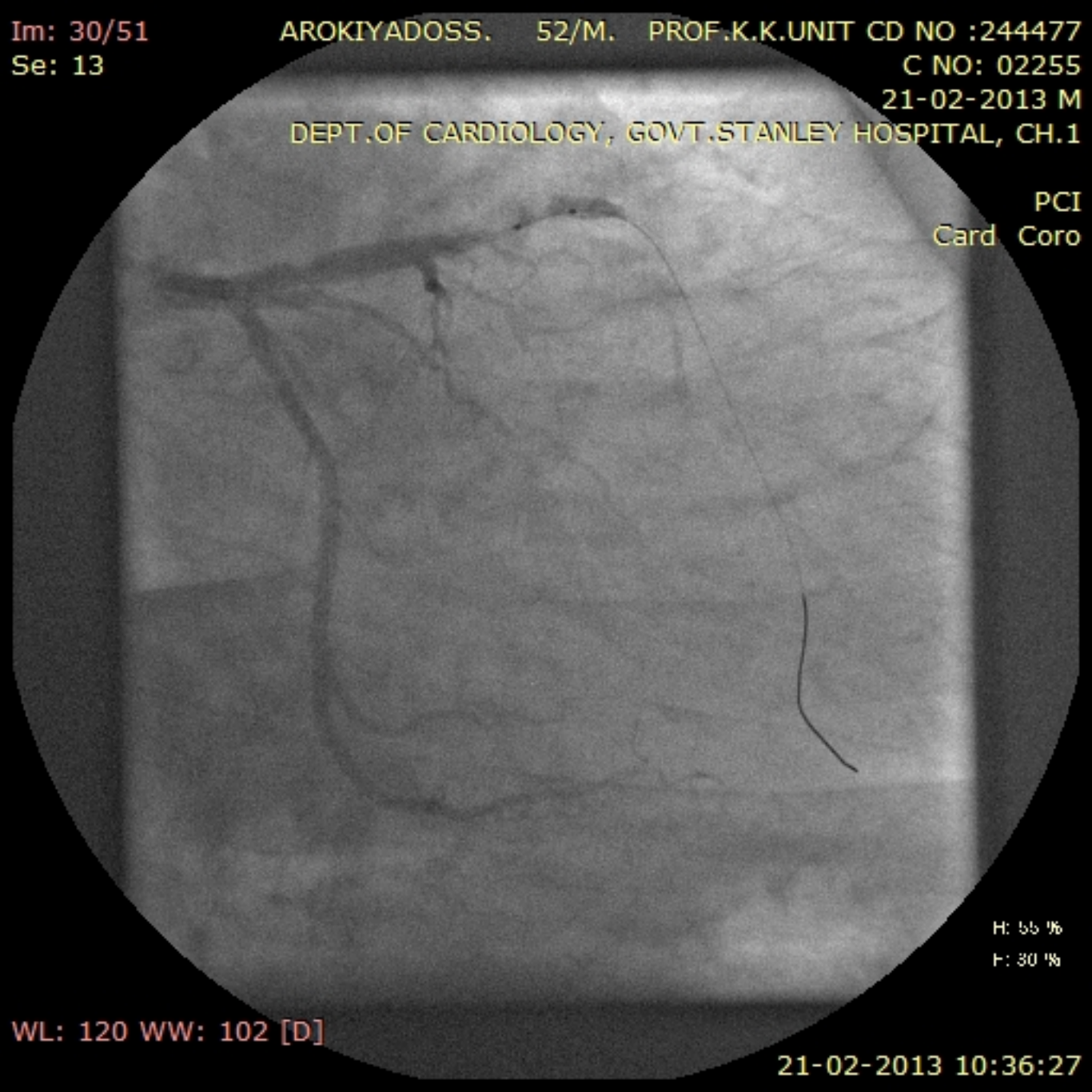
12 Lead; Standard Placement

Unconfirmed Diagnosis




Device: Speed: 25 mm/sec Limb: 10 mm/mV Chest: 10.0 mm/mV F 50~ 0.50-100 Hz W PH100B CL P?


H: 55 %
F: 30 %


Im: 30/51
Se: 13
AROKIYADOSS. 52/M. PROF.K.K.UNIT CD NO :244477
C NO: 02255
21-02-2013 M
DEPT.OF CARDIOLOGY, GOVT.STANLEY HOSPITAL, CH.1
PCI
Card Coro
H: 55 %
F: 30 %
WL: 120 WW: 102 [D]
21-02-2013 10:36:27


H: 55 %
F: 30 %